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ON THE VISCERAL MANIFESTATIONS OF THE ERYTHEMA GROUP OF SKIN DISEASES.

[THIRD PAPER.]

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According to the classification of recent writers under the erythemas—les erythèmes of the French—are included simple erythema, erythema exudativum, herpes iris, erythema nodosum, certain of the purpuras, urticaria, and angioneurotic œdema. The essential process is a vascular change with exudate, blood, serum, alone or combined. While five or six of the affections just named are described usually as separate diseases they belong to one family, and are characterized by the similarity of the conditions under which they occur, the frequency with which the lesions are substituted the one for the other in the same patient at different times, the tendency to recurrence, often through a long period of years, and, lastly, the identity of the visceral manifestations. In the latter for some years past I have been particularly interested, and in 1895 I published a series of eleven cases,[1] and in 1900[2] a second series of seven cases. In this paper I shall give a third set of eleven cases, and shall consider the features presented by the entire group of twenty-nine cases.

Eight only of the patients were under ten years of age; thirteen were between the tenth and twentieth years. There were eleven females and eighteen males.

The seriousness of the condition is attested by the occurrence of seven deaths in my series—24.1 per cent.

[1] The American Journal of the Medical Sciences.
[2] Jacobi Festschrift and British Journal of Dermatology, vol. xii.

CASE XIX. *Onset, with erythema over cheeks and nose, in October, 1889; puffiness of one small joint; œdema of one eyelid; increase in the erythema and swelling of the face; marked puffiness and stiffness of the hands; recurring attacks of erythema of the face and local infiltrations in different parts of the body; slight fever; in March very high fever, to 105° F.; much swelling and infiltration of the face and neck; acute nephritis; persistence of œdema of face; death by uræmia May 13, 1900.*—Miss C., Pittsburg, Pa., aged fifteen years; seen January 9, 1900. Father neurotic. No rheumatic history in the family. She has been a very healthy girl; is well grown. She began to menstruate at fourteen years. Large framed; has developed rapidly. Late in October, one Sunday returning from church, an erythematous blush of both cheeks was noticed, extending to the bridge of the nose. It was thought to be due to the sun, as it was a very hot day. As the congested condition of the face did not disappear, a dermatologist was consulted, who pronounced it eczema and gave her an application. For two weeks subsequently she showed a slight puffiness over the tendon of the left metacarpal bone of the middle finger. This disappeared quickly, then followed a puffiness of the left eyelid, and in a few days of the right eyelid, the erythema of the face changing in appearance, at times nearly fading. The eyelids were infiltrated and œdematous (not red). The urine was repeatedly examined and was negative.

Just before Christmas the face became very much more swollen and the eyes were nearly closed. The erythema became much more intense. On Christmas-day the backs of both hands over the middle metacarpal regions were very puffy and œdematous (not reddened). There had been no itching or burning of the face or hands. She had one attack of pain under the left breast—pleurodynia, the physician thought—quite severe, relieved by three days' rest and local applications.

Present Condition. The patient looks remarkably well.

Face. There is a marked erythema, with swelling of both cheeks and the nose, and to a less extent over the chin. It had a purplish-red look as she came in from the cold air. After rest in a warm room it became of a brighter red. On the cheeks it extends to within about 1½ cm. of the angle of the mouth, a little more extensive on the left than on the right. The nasolabial fold, the whole of the under lip, and the greater part of the upper lip are uninvolved. The nose is involved, about the same color as the cheeks, and a slight extension can be seen over the supraorbital ridges, a swelling and a slight blush of redness. The eyelids are at present only a little puffy; the conjunctivæ are slightly congested; on the chin the patches are discrete; individual isolated spots, slightly infiltrated and a little raised, and extend under the chin and behind the ears. There is no ecchymosis. The tonsils and pharynx look natural.

The left hand feels stiff, and to make a fist is painful. On the back of this hand, during Christmas-week, there was a large serous infiltration of a few hours' duration. There is now only a slight patch of erythema over the second metacarpal bone of the middle finger. On the back of the right hand there are a few scattered patches. On the back of the fingers of the right hand there are raised reddened patches, the largest one on the index.

Dr. Duhring, of Philadelphia, saw the patient the next day, and regarded the rash as a peculiar form of erythema.

The subsequent history of the case is as follows:

On February 6th the doctor wrote: "The lobes of the ears have desquamated, leaving a normal appearance, except a little dry, thin scale, not yet loosened; the redness and puffiness of the lobes have disappeared. The upper outer angles of the redness of the cheeks show a tendency to desquamate; a little roughness of the skin; thin, scaly epidermis. The upper lip, which was blotchy, has desquamated, the base being still red. The contour of the face is improved; the general erythema of the cheeks varies. The œdemas and infiltrations change from point to point: upper eyelids slight, one elbow from time to time, both knees or one knee, a finger or the back of the hand. On Sunday afternoon it developed beneath the tendo Achillis of left foot; previous to that both ankles on their outer aspects; last night the left ankle, the right knee, with some pain, and the right elbow was so stiff that it was difficult for her to use her knife or fork with that hand at dinner. Occasionally, when the œdema is more prominent, there may be a feeling of heat to the touch, the temperature at bedtime ranging from 99.8° to 100° F., to 100.2° F., to 100.4° F.; at other times normal, morning and evening. The œdema lasts from twenty-four to thirty-six hours, the back of the left hand week before last proving to be the most persistent. This morning in bed she had free movement of all her joints, showing that the disturbance of yesterday was passing away. As a rule, toward each evening she feels stiff, it may be in the extremities or hands, but by morning, unless the œdema is prominent, the stiffness is not noticeable. The spaces beneath the quadriceps tendon, the tendo Achillis, and beneath the malleoli of the ankles, and the metacarpals are the points now where the swellings occur. The eyelids have never been so bad as during Christmas holidays. Locally the face looks badly enough. Physically, notwithstanding the recurring œdema, I consider her better than one month ago. In animation and general good spirits she is becoming more natural, though the going out among people requires some pluck and determination."

On February 19th I saw the patient in Pittsburg. She seemed somewhat better, but she had still the swelling of the face and of the ear, and occasionally erythematous patches over the nose. For a few days she had had a little fever, up to 100° F., and the day I saw her she was not feeling so well and the temperature had risen to 103° F. The spleen was palpable. Then for a month she had a very severe attack of extensive inflammation of the skin of the face. She had high, irregular fever, ranging to 105° F., and the doctor writes: "The force of the trouble, whatever the cause, expended its power on the face and neck. No clinical picture could compare with it in my experience, except that seen in erysipelas—I mean in dimension, appearance, and parts involved. The extremities have remained perfectly free. The submaxillary and sublingual glands—in fact, the entire circumference of the neck was distended. The infiltration was so extensive that on March 2d she complained of pain in the left ear, and on March 5th the right ear was in the same condition, marked dulness of hearing, and on March 14th paracentesis of the drum had to be performed. The drainage relieved her." The temperature did not begin to fall until after March 20th.

On April 10th the doctor wrote: "Her condition is no worse than when you last heard. The pathological process, whatever it is, is con-

fined to the face and forehead. The rise and fall in the morning and evening temperature keeps about the same, varying from 99.8° to 100° F. The glands of the neck are not so large. There is some œdema of the parts. For several days in the late afternoon she is having a pretty severe frontal headache, which prostrates her. Her appetite is good, and she sleeps well. She sits up a little every day, but soon tires. She is taking a dessertspoonful of Basham's mixture with a sixtieth of strychnine. I have been hoping that no more serious complications might arise, but it is evident that we are not at the real cause of the trouble. The report of the urine is as follows: specific gravity, 1016; considerable amount of albumin, a few red blood corpuscles, hyaline and waxy casts.

"Since you saw her there has been but one outbreak of the œdema such as appeared at the beginning; it came on the back part of the left hand last week, and persisted for twenty-four hours."

On May 17th the doctor wrote: "During the latter part of March and the first part of April she was gaining. On Easter evening there was a rise of temperature, the albumin increased rapidly, and nausea and vomiting became very distressing. She had perforation of the drum of the right ear. The œdema of the face persisted. During the last week of life the left eye was closed entirely and the left half of the face was tense with œdema. The amount of urine diminished to two and a half ounces in the twenty-four hours. The mind kept clear. She died of uræmia on Sunday, May 13, 1900."

This case and No. XXVI., which presented many points of similarity, illustrate the very grave character of some of these obscure forms of erythema. Here was a healthy young girl of good family history who had a persistent erythema of the face and hands, with areas of angio-neurotic œdema; then three months later a more severe attack, with fever and intense swelling of the face and an acute nephritis, which proved fatal seven months after the first appearance of the erythema.

CASE XX. *Attack of severe colic; admission to the surgical side for suspected appendicitis; a week before a purpuric eruption on the legs, with high-colored urine; rash of exudative erythema on the legs, with here and there purpura; no involvement of the joints; acute nephritis, abundant albumin, and blood casts, and a small amount of blood in the stools; recovery.*—This case is particularly interesting on account of the fact that he was admitted to the surgical side of the hospital with abdominal symptoms, which were suspected to be due to appendicitis.

Milton R., aged fifteen years, admitted March 14, 1900. He had been a healthy boy; never had rheumatism; no rheumatism in his family. He had never had similar attacks, though his mother said he had irregular, colicky pains at times, and once of such severity that he vomited with it.

Present Illness. About March 7th he noticed himself that the urine was very dark. He had had, a week or so before, some colicky pains in the abdomen.

With the highly colored urine there was no pain in the back, micturition was not painful, and there was no increased frequency. About the same time it was noticed that his legs were somewhat swollen and

peppered with red spots, which continued to come out at intervals. The boy had been going to school and had seemed quite well up to this attack.

On admission he was a well-nourished, healthy-looking boy. There was no rash upon the face. Symmetrically arranged on the arms and elbows and about the wrists and hands were a few patches of a rash, chiefly discrete, consisting of papules 1 to 3 mm. in diameter, some distinctly raised, many of them capped with small vesicles, which in places were drying. About the elbows the patches were larger, more raised, and had a purplish-red color. They varied in size from a pin's head to 5 cm. in diameter. In the larger areas the centres were more yellowish in color, the periphery purplish red. The color did not entirely disappear on pressure.

On the legs there was an extensive symmetrical rash about the ankles and knees, the patches very diffuse, on the legs more discrete. On the outer sides of the legs there were patches 3 by 1.5 cm., slightly raised, and these presented three grades of color, the centre whitish, a reddish-brown staining, and then a zone of brighter red. Scattered between the larger areas were some distinct purpuric spots.

Heart. Apex-beat in fifth, 7 cm. from midsternal line, rather diffuse, no thrill, soft apex systolic murmur heard over the body of the heart.

The abdomen looked natural, soft to touch; liver not enlarged; spleen slightly enlarged, the edge distinctly palpable below costal margin. Slight general enlargement of lymphatic glands; reflexes normal.

The urine on admission was blood-red, specific gravity 1014; contained many granular casts, large and small, and some red blood casts; abundant albumin. I remarked to the class the first day how interesting it was to see a case of acute hemorrhagic nephritis without any diminution in the amount of urine, with no swelling of the face, and no trace of œdema. The temperature was 100.5° F.

Blood. Hæmoglobin, 60 per cent.; red blood corpuscles, 4,156,000; leukocytes, 6800; coagulation time, two minutes and a quarter. On March 3d blood cultures were taken, which were negative.

The rash gradually faded and no fresh spots of exudative erythema appeared, but on several days between the 18th of March and the 1st of April small purpuric spots appeared, some minute ones on the palms of the hands.

On March 18th the patient had a small stool containing clear mucus and bright-red blood.

The boy progressed very favorably. The urine on the 17th was 1720 c.c. The bright-red color persisted, and began to clear about March 22d. It remained smoky and had blood in it up to the middle of May. The albumin gradually disappeared. Red blood corpuscles, hyaline and granular casts were seen all through April.

On May 11th the condition was as follows: smoky, 1013; acid reaction, trace of albumin, granular casts, red blood corpuscles, red blood-celled casts. The patient was reported as quite well in the autumn.

CASE XXI. *Otitis; swelling of the hands and legs; extensive purpuric rash, in places papular; acute nephritis, with albumin, tube casts, and blood; cramps in the abdomen and vomiting; recovery; subsequent admission with meningitis from the otitis media; death.*—John D., aged twelve years; seen April 24, 1900. He complained of running from the left ear

and swelling of the hands. His family history was good; no similar troubles that he knows of; no rheumatism. Except summer complaint when he was two years old, he has been an exceptionally strong, healthy boy; has had no acute infectious diseases. About two weeks before he applied at the Dispensary he had a running from the left ear. Five days before application his hands and legs became very much swollen, and he could not put on his shoes. The day before application a rash came out on the legs.

Present Condition. Fairly well-nourished boy, but he looked anæmic. The face and eyelids were somewhat puffy. Evidence of recent epistaxis about the nostrils. The hands looked swollen and œdematous. The pulse was rapid, regular. There was no enlargement of the heart. The first sound was rather thudding, and the second pulmonic was accentuated. Temperature, 101° F. The abdomen looked full; the spleen was easily palpable. The liver was not enlarged.

Below the knees the legs were much swollen, firm, and did not pit on pressure. There was an extensive petechial rash, papular in places, but nowhere with much swelling. The urine showed albumin and coarsely granular tube casts; no red blood corpuscles. He was ordered an iron mixture.

April 27th. The patient came again to-day; says he feels very much better. The œdema of the legs has almost gone and the rash has faded. The urine shows a' .ndant albumin, numerous coarsely and finely granular and hyali'.e casts.

May 1st. Since the previous note the patient has had recurring attacks of severe cramps in the abdomen, with vomiting. The mother describes the material brought up as at first bloody, then black, and later yellow. The swelling of the face and feet is almost gone.

4th. Patient has vomited every evening since the last note; has not vomited to-day; does not complain of any pain.

10th. Patient began to vomit on waking, and continued to do so at intervals during the morning.

12th. He has had no nausea or vomiting since the last note. The legs are not œdematous. A few stains of the purpura remain.

14th. The urine is light colored, cloudy, 1 per cent. of albumin by Esbach, and contains a large number of red blood corpuscles, finely granular casts, and casts of red blood cells.

On July 30th the boy was admitted to the hospital with acute meningitis from the otitis media. He was removed by his parents on August 1st, and he died a few days later.

CASE XXII. *Swelling of the legs, with purpura and pains in the knees and ankles; colic; acute nephritis, with blood, albumin, and tube casts in the urine; recurring attacks of purpura, with urticarial wheals; recovery.*—Edna C., aged six years; seen May 11, 1900, with Dr. Richardson, of Bel Air. Family history good. The child had been well and strong; never had rheumatism or chorea. Five weeks ago, about 4 P.M., the child complained of pains in the legs, and the mother noticed that they were swollen, and dark blotches came out. The legs became swollen as far as the knees, and it was difficult for her to move the knees and ankles. She has had for a year or so slight colicky pains in the stomach. On the following day Dr. Richardson found a good deal of albumin in the urine. On the day of the attack the urine contained blood. She has got pale, has had several recurrences of the

blotches on the legs, and has with it much colic. Yesterday she had a very severe attack.

She is a fairly well-grown child; looks a little pale and brown, partly sunburn. Color of the lips pretty good. No swelling of the eyelids; no puffiness of the wrists. The tongue is clean; no swelling of the joints. There is a fresh crop of blotches, which came the night before last. The original crop was more than this. At present there are large areas 2 cm. across, slightly raised, evidently fading spots of urticaria, with purpura. Then there are in addition spots of simple purpura scattered between these large areas. The purpura extends as far as the hips. The spleen is not palpable, liver not enlarged. The apex-beat is within the nipple line, no thrill; heart sounds are clear. The urine has been albuminous at times.

The urine to-day had a specific gravity of 1024, clear, acid reaction; contained neither albumin, sugar, casts, nor red blood corpuscles.

March 13, 1903, the mother writes that there has been no recurrence of the purpura.

CASE XXIII. *History of attacks of pain in the knees; colicky pains, with nausea and vomiting and diarrhœa for many years, sometimes with swelling of the feet; enlargement of the spleen; recurring attacks of purpura, with abdominal pain and vomiting; outbreaks of erythema; recovery.*—Sarah E. W., aged fourteen years, admitted September 10, 1900 (Med. No. 11859), complaining of stomach trouble. Her mother had had rheumatism. She herself has an indefinite history of rheumatism, irregular pains, and has had three or four attacks in which her knees have been painful; no swelling, no redness, no fever. She had been subject to sore throat, especially three years ago. At times she has had attacks of vomiting. The child's mother states that she was well and natural until the attack of measles at her fourth month. Ever since she has had two serious troubles: first, attacks of abdominal pain, with nausea, vomiting, and diarrhœa; and, secondly, skin rashes, which come out suddenly and then fade away. They are usually symmetrical. With these attacks there is often swelling of the feet, which comes very rapidly.

For three weeks she has been having pains in the abdomen, and for five days past there has been much nausea and vomiting, and three days ago she had diarrhœa, five or six stools a day. She felt feverish, had some sweats at night, and loss of appetite. She has had to get up at night once or twice to pass urine. Eight days ago the feet and legs began to swell, and a red rash broke out on the skin. The knees were painful, and her eyes at the same time became puffy. The swelling of the legs has disappeared and she has been a good deal better.

On admission she was a small, pale, frail-looking child, not anæmic, eyelids slightly puffy. The temperature was 100° F.; pulse 116, slightly irregular. There was a somewhat diffuse heart-beat in third, fourth, and fifth; on palpation a systolic thrill; on auscultation a blowing systolic bruit transmitted to the axilla; accentuation of the pulmonic second. The abdomen looked natural; the liver was not enlarged; the spleen was fully 2 cm. below the costal margin. There was slight œdema of the feet and ankles, and there was a fading rash on the knees, elbows, over the clavicles and ankles. Some of these spots were distinctly purpuric. One above the left external malleolus was a bluish spot, looking like a bruise.

September 13*th*. Had an attack, with great coldness of the feet and a burning sensation in the pit of the stomach, which increased so that at 10 P.M. she had a great deal of abdominal distress. A fresh, brilliant-red rash in raised patches came out on both buttocks, extending down the thighs, and on both elbows. The abdominal pain persisted that night, and at 6 A.M. she vomited, at first curds, later a greenish fluid, and about 9 o'clock mucus mixed with blood.

14*th*. There is this morning an abundant purpuric rash over both buttocks, extending down the thighs, up the back on each side, over the elbows, and the upper arms. The rash is raised, and the individual spots have distinct margins. On the right elbow there is a bluish-red discoloration, which is painful to the touch. There is no pain or swelling in the joint itself. She had to have morphine. Throughout the day she vomited several times.

Blood. Red blood corpuscles, 3,680,000; leukocytes, 10,000; hæmoglobin, 75 per cent.; blood coagulation time, four minutes.

Urine. Specific gravity, 1012; no albumin, no blood, no tube casts.

15*th*. The rash is very extensive over the back. Some of the purpuric areas are infiltrated and raised and measure 5 x 3 cm. There is a little swelling in the left elbow-joint to-day. There are red blotches on the thumbs and over the knuckles. The spot where the hypodermic needle was inserted is surrounded by a hemorrhage.

16*th*. The swelling of the left elbow has disappeared. There is a fresh erythema on both forearms. From this time the patient improved. There was a good deal of pigmentation and staining left by the hemorrhages. She was discharged, feeling quite well, on September 24th.

CASE XXIV. *Erythema and purpura; acute nephritis; general œdema; persistence of the albumin for two months; gradual recovery.*—Morris S., aged three years, Hebrew; admitted December 20, 1900, with swelling of the face, abdomen, penis, and legs. There was nothing of any moment in his family history; no rheumatism. He was healthy as a baby; had a sore navel for some time. When two years old he had diphtheria, a severe attack, not followed by any swelling of the feet. With this exception the child has been very well. He has grown and thriven, eats heartily, and drinks three large cups of coffee in the day! Three days ago the father noticed some red spots on the back of the left hand, which looked like bites of insects. At the same time there was swelling of the wrists and back of the neck. That night the swelling became worse and the spots extended and formed large blotches. The father said the wrists looked as if they had been scalded. On the following day the spots appeared on the legs and body and became darker. The penis became œdematous.

I saw the child at my out-patient clinic and made the following note: There is well-marked purpura over the legs and around the crests of the ilia, and there is a general subcutaneous œdema of a brawny character. The skin of the penis is œdematous. The swelling and œdema which had been so marked on the wrists has disappeared and has left a brownish-red stain. The abdomen is large; there is dulness in the flanks; no percussion wave. Some œdema of the lower part of the back. On the outer surface of the skin of the right arm there are stains of a purpuric rash. Examination of the urine showed a large amount of albumin, but no tube casts.

The child was taken home by its father the same afternoon, but returned three days later with general anasarca and signs of slight fluid in the abdomen. The child looked very well. The face was only slightly swollen, the eyelids a little puffy. The urine had a specific gravity of 1023, dark yellow in color, and there were a few casts and much albumin. Throughout the month the dropsy persisted to a slight extent, but gradually disappeared. A very careful daily study of the urine was made, particularly with reference to the percentage of albumin and urea. The amount of albumin varied through January from 0.1 to 0.3 per cent. On the 17th, 18th, and 19th of February the amount increased rapidly, reaching on the 20th 1 per cent. There were no tube casts. On March 5th, for the first time, the urine was free from albumin. Then again, from the 25th to the 30th, the albumin returned, and reached on the 28th 0.5 per cent. From that time on the albumin was absent, and he was discharged April 21st in good condition. I heard on March 1, 1902, that the patient has remained perfectly well.

CASE XXV. *For the first six years of life recurring attacks like nettle-rash; then to the tenth year freedom; in tenth and eleventh years recurrence of the rash in larger blotches and much swelling of the face and eyes; recurring attacks of severe spasmodic croup, having no special relation to the skin rash; twice with these attacks there has been swelling of the throat; lately marked swelling of the soft palate and uvula; swelling of the feet and of the face; no nephritis.*—N. D. D., aged fourteen years, Lafayette, Ala.; sent May 6, 1901, by Dr. Love. When three weeks old red splotches came out all over him and itched like nettle-rash; lasted three weeks. It recurred periodically until he was six years old. He never passed six months without an attack; sometimes they would occur quite frequently. He had no pain in the abdomen with the attacks, no vomiting. From the age of six to ten years he was perfectly free from the attacks. At ten years the trouble reappeared, and recurred periodically until he was eleven years old. Good health until the thirteenth year, when the disease returned. The "splotches" were larger and there was swelling of the eyelids and face. About six weeks ago the inside of the throat began to swell, chiefly the uvula and the soft palate, and there was œdema of the outside of the neck. He has had one attack also in the left auditory canal. The last attack was ten days ago. The feet began to swell and he could scarcely get his boots on. The face was a little swollen in this attack.

The family history is good. The father has a quinine idiosyncrasy, and cannot take even a few grains without having a skin rash. An uncle also has the same peculiarity.

He is a healthy-looking boy, of good color. No swelling of the face to-day. The remnant of an urticarial spot just above the right eyebrow. The only eruption at present is above the popliteal space on the left side, where there is a large spot extending across the lower third of the thigh, measuring 8 x 10 cm. It is reddened, prominent, greatly infiltrated, the margins localized and distinct; a dusky red color, in places of a more vivid red; very hot. In one or two places the margins look a little less red. The hyperæmia is extreme, and pressure fills up at once. There is nothing at present in his throat; no other spots anywhere.

The heart sounds are clear; arteries not sclerotic. Pulse is good. Hæmoglobin, 80 per cent. Urine: no albumin, blood, or tube casts.

All through his life he has been subject in the winter to attacks of severe spasmodic croup, which have not apparently had any relation to the skin rash. It has recurred at intervals all his life, twice with marked swelling of the throat. It commences with difficulty in breathing, and lasts twenty or thirty minutes. He will call and say he is about to choke. Wet cloths about the throat relieve it. No cough with it.

May 8th. The swelling in the patch of the day before yesterday has subsided. There is left a bluish-green stain like a bruise.

I heard from Dr. Love on February 23, 1903. The boy is very much better. He has had no attack for two months. He took calcium chloride for a long time with benefit; recently camphor in three-grain doses four times a day, which seems to have had a very satisfactory influence.

CASE XXVI. *Onset in September*, 1901, *with erythema of the nose and cheeks; extension to the elbows and arms, usually in the form of wheals, but some spots purpuric; chill, followed by consolidation of the lower lobe of left lung; protracted fever; enlargement of the lymphatic glands; delayed resolution of the pneumonia; urine clear in the attack; gradual recovery; in May*, 1902, *onset of acute nephritis; uræmia; death in a convulsion.*—L. E., aged twenty-four years; seen at Pittsburg, Pa., on February 6, 1902, with Dr. Litchfield. Her family history was good; no rheumatism; no tuberculosis. She had been a very robust girl; no serious illnesses. Toward the end of the summer, when at the Thousand Islands, a rash began on the face, chiefly on the nose and cheeks. It looked, the doctor said, like lupus erythematosus. It persisted, and troubled her a great deal. Dr. Fox, of New York, who only saw her once, writes that he thought it looked like an acute lupus erythematosus. It spread in patches to the body, particularly about the elbows and on the arms. The patches were red, sometimes raised, and would, at times, come out like wheals. Before Christmas she had a very violent outbreak, and many of the spots became purpuric, and on the arm the doctor said there were spots which looked almost like the eruption of vaccinia. On December 27th she had a chill, and on the third day consolidation of the left lower lobe was found. The temperature was 104° F. From the fifth and sixth days there was a drop in the temperature. On the twelfth day it dropped to 98° F.; then it gradually rose, and from the twelfth to the twenty-sixth day it ranged to 105° F. Then the other lung became involved. There was very little cough, particularly after antipneumococcic serum was given. There were no true chills. The mind was clear, as a rule. For the past three weeks the temperature has ranged about 101° F., with no very great drops. The pulse has been from 112 to 120; respirations 22 to 40; mind clear; no delirium. The right lung has been clearing. The left lower lobe has been solid for some weeks. About two weeks ago the left leg became swollen, and she had a thrombus in the femoral vein. For ten days the lymphatic glands throughout the body have peen a little enlarged and tender.

At the time of my visit the patient looked thin and pale. There was no sign of any rash on the face, but there was erythema of the skin of the thighs, mottled and patchy, and in places a little raised. There was no purpura. The left leg was swollen, but was no longer tender. She had complete consolidation of the lower lobe of the left lung, with tubular breathing and a few râles. The fremitus was not very marked.

I put in a needle in two places, but got nothing. The Widal reaction was negative. The sputum was a little rusty, and I had some examined. There were pneumococci, but no tubercle bacilli.

The urine was examined repeatedly, and there was occasionally a faint trace of albumin, but nothing special. She was anæmic, and on January 29th blood count showed: red blood corpuscles, 3,310,000; hæmoglobin, 46 per cent.; leukocytes, 6000. The patient gradually improved through March and April, and seemed to be getting quite well. In May she went to Atlantic City, where she improved rapidly. Toward the end of the month an acute nephritis came on, without any special exposure; the urine was scanty, high colored, contained blood and tube casts and much albumin. There was fever, 101° to 102° F.; no skin rash. I saw her with Dr. Marvel shortly after she had had a uræmic convulsion. She died within a week of the onset of the nephritis.

CASE XXVII. *From infancy recurring attacks of colic, sometimes with vomiting; recurring attacks of blotches (erythema) on the skin of face and arms; at the time of examination a spot of erythema capped with blister.*—Dorothy B., aged thirteen years, referred to me by Dr. Charlton, of Savannah, Ga., June 18, 1902, for a peculiar trouble in her stomach. She was healthy when born, was a healthy child, and has grown and developed well. The family history is good; no special peculiarities. Since infancy she has had a peculiar stomach trouble, recurring attacks of nausea, and colic-like pain. The attacks recur at irregular intervals, a week or two apart, sometimes a number in succession, and then she will be free for several weeks. She will appear perfectly well, will eat a good meal, and an hour or so afterward will say: "May I go and lie down? I have a pain in my stomach." The attacks are chiefly in the morning. The abdomen seems tender and the colic at times has been severe enough to make her cry out. It has been a source of constant worry, and nothing has ever seemed to do her any good. At times she has had very obstinate vomiting with the attacks. Her mother says the pain is always in the pit of the stomach. When four or five years of age she had a very bad attack of "ivy poisoning." This spring some red spots came out on the neck, and she has had them recurring ever since. They occur on the face and arms as red blotches, and then become capped with little blisters.

The patient is a healthy-looking girl; color good; tongue clean. The abdomen is soft, natural looking; spleen not enlarged; stomach not dilated; no enlargement of the liver. No sensitiveness anywhere on pressure. The heart is not enlarged; the first and second sounds are loud. There is a soft systolic murmur at the base. The skin seems everywhere clear, except on the left foot, just above the middle toe. Here there is a raised, infiltrated, reddened spot about 5 x 5 mm., capped with a small, clear blister. This, her mother says, is the typical appearance of the rash. When the attacks are bad a number of these appear on the face and neck, and they come out on the extremities, looking at first like hives, and then are capped by the little blisters. The urine is clear; no blood.

A letter from her mother, March 22, 1903, states that the child has improved very greatly, and has been very free from that colic, and has had no return of the skin trouble.

CASE XXVIII. *For two years recurring attacks of purpura, chiefly on the legs; severe attack three months before admission, with colicky*

pains, vomiting, diarrhœa, and arthritis; in the last attack diarrhœa was a marked feature, with the passage of blood from the bowel.—A. S. A., aged twenty-six years, white, single, lawyer, Montgomery, W. Va.; admitted February 12, 1902, complaining of skin disease and stomach trouble. No similar trouble in family; no purpura. Patient's father has chronic diarrhœa. Usual diseases of childhood. Typhoid fever in 1898, slight attack, but followed by a gastric (enteric?) trouble lasting about four months. Patient was in bed altogether about six months. He has had occasionally swollen and tender joints. One attack of swelling and pain about tendo Achillis. Appetite and digestion good prior to present illness. Tendency to constipation. Denies lues and gonorrhœa. Habits good. Average weight, 160 pounds. Present weight, 140 pounds. Patient first noticed the purpuric eruption on his legs in the spring of 1901, two years ago. At this time the spots were very small and situated only below the knees, especially about the ankles. At this time the patient had no nausea, vomiting, or diarrhœa; no arthritis. During the summer of 1901 the patient had indigestion and constant recurrences of purpura; no arthritis; some nausea, vomiting, and pain. At no time was he confined to his bed or compelled to give up his work.

In December, 1901, the patient had his first severe attack, which kept him in bed for about a week, and was soon followed by another of about the same severity and duration.

For eleven months, until November, 1902, the patient was well; then he had very severe abdominal pain, with diarrhœa, nausea, and vomiting, followed by arthritis and a hemorrhagic eruption on his legs. He was forced to give up work and go to bed, and until New Year's there were recurring attacks. During the last month or six weeks he has been at work, but has been feeling badly, with arthritis and purpura, some "indigestion," and pain.

The attacks are all of the same character. The patient first noticed the purpura on his legs or arms (at first it was only on his legs below knees, but is now on thighs above the knees, and on hands and arms, particularly on extensor surfaces), accompanied with a mild arthritis. The joints are "sore and stiff," but he can use them. This is followed by a severe pain in his left side and left half of abdomen, passing over middle line in a narrow band. The pain is fixed, not radiating, and of a cramp-like character; sometimes requiring morphine by the mouth or hypodermically for relief. The pain usually lasts about two days. Following the pain the patient has diarrhœa and usually nausea and vomiting. The vomiting is the last event in the series, and about the time it begins the purpura fades, leaving brownish-yellow stains.

The joints attacked are usually the ankles and knees. In the last outbreak the left hand (metacarpophalangeal joints) was involved, and his wrists, never his elbows, shoulders, or hips. Last November the patient passed considerable blood per rectum of a very dark color. He has never had hæmatemesis or hæmoptysis. Slight epistaxis. No hæmaturia or bleeding from gums or mucous membranes. He has grown quite pale in each attack, and loses flesh rapidly, but regains it quickly.

Present Condition. Well nourished, but pale. Examination of heart, lungs, and abdomen negative. There is a purpuric eruption on back of hands and arms, more on right arm than on left, and on extensor than on flexor surfaces; same on feet and legs up to the groins. Tender-

ness and subjective feeling of stiffness in left hand (metacarpophalangeal joints) and right wrist. Blood pressure, 145 mm. of Hg. Red blood corpuscles, 4,280,000; leukocytes, 7600; hæmoglobin, 65 per cent. (von Fl.). Blood coagulation time, six and a half minutes. Urine negative.

On February 13, 1902, I made the following note: Patient is pale, sweating profusely. Tongue furred. Skin of face clear. Gums not swollen; a little red line; no blue line. There is a fading purpura on the arms; spots not raised, brownish, leaving stains, chiefly on flexor surface. No purpura on trunk and back. On the legs there are numerous stains of fading purpura over feet and extensor surfaces. The spots are larger on the legs. All are now fading. Joints nowhere red or swollen.

Circulatory System. Slight sclerosis of arteries. Apex beat visible in fourth left interspace, just inside nipple. Soft murmur at apex with first sound and over body of heart, not propagated to axilla.

Abdomen looks natural; nowhere painful; a little tender in spots over colon. Spleen not enlarged. Liver not enlarged. Appendix not palpable. Superficial glands not enlarged. He was ordered calcium chloride.

February 14th. Blood coagulation time, six and a half minutes.

16th. Purpura has almost disappeared. Patient has improved very rapidly. On his discharge a few days later the patient looked very well; blood coagulation time was three minutes.

Case XXIX. *Acute otitis; arthritis; recurring attacks of colic, with occasional vomiting; hemorrhage from the bowels; purpura; acute nephritis; recovery.*—March 7, 1903, E. F., aged five years, seen with Dr. Hamburger, to whom I am indebted for the notes.

Personal History. He had a prolonged attack of summer diarrhœa two years ago. It followed an attack of otitis. For the past two weeks he has had pain in the ears. Last Wednesday (to-day being Saturday) there was a discharge from both ears. Fever has persisted off and on since the onset. This morning he was unable to stand on his feet because of pain. Temperature, 100.5° F. There is a purulent discharge from the right ear. Heart sounds are clear. Examination of the lungs negative. The spleen is not palpable. There is no tenderness in the abdomen. The dorsum of the right foot is swollen, not reddened, but very tender on palpation. There is some tenderness, but no swelling over the left foot.

March 8th. The patient had a restless night. His temperature rose to 102° F.; this morning it is 99.7° F.; pulse, 104. He has complained of pain in the wrists and in the interval between the thumbs and first fingers. He has pain in the abdomen, accompanied by vomiting of the milk. Heart sounds are clear.

9th. The patient was unable to retain food yesterday, and he has vomited this morning the nourishment, as well as some bile independent of taking food. Throughout the night he has cried because of intermittent pain in the abdomen, referred now to the left half and to the umbilical region. His temperature this morning is 99.6° F. The tongue is heavily coated. There is a decided fœtor oris. Every few moments he cries out with pain in the abdomen, which lasts a few minutes, then disappears, only to return in from ten to twenty minutes. He rolls over in the dorsal decubitus in these paroxysms. The joints are no longer sensitive. The abdomen looks natural. The left half is sensitive on

palpation, particularly in the iliac region, while above in the upper half a fine gurgling can be detected. The abdomen is exquisitely tender during the painful period. Following an enema, a quantity of blood, sufficient to discolor the water a deep, dark red, was ejected with some fecal material. Early this morning he had a small movement (a couple of scybalæ) without blood. For the past four or five days the ears have been irrigated with 1.44 of bichloride of mercury to one quart of water three times a day.

10*th*. The boy had considerable pain during the night. Yesterday afternoon about 3.30 he passed about one ounce of pure blood. This morning he is comfortable. Pulse, 120; temperature, 98° F. The tongue is coated, but it is clearing at the tip and sides. There is no gurgling over the abdomen. There are no petechiæ. He is being fed on albumin water, and toward evening is given a starch and laudanum enema.

11*th*. The pain returned about noon yesterday. It was so intense that at 5.30 P.M. the starch and laudanum enemas were resumed. He had two stools, composed mainly of blood and in part of the enema. The night was restlessly spent in pain. Early in the morning he passed a bloody stool. The temperature this morning was 97.8° F.; pulse, 108. To-night the temperature is 98.6° F.; pulse, 93. The day was relatively comfortable and free from pain, but this afternoon about 6.30 the colic returned with such intensity that the patient cries out. There is no skin eruption. The starch and laudanum enemas are being discontinued and he is being given paregoric, half-ounce, every two hours.

12*th*. The colic continues this morning. He has had two stools to-day, one being composed of narrow, long casts, dark brown in color, evidently composed of changed blood. The other stool contains curded milk, blood, a little mucus and some fecal matter.

Urine acid, 1026, yellow, no albumin, no sugar.

13*th*. He had a great deal of pain during the night. This morning at ten he was given one-half ounce of castor oil. At 3.20 P.M. he had a stool approaching the normal. To-night another movement of blood and fecal matter. The day was quite comfortable, but beginning at about 8.30 the colic returned. Paregoric, twenty drops, and deodorized tincture of opium every two hours.

14*th*. The boy was very restless until 3 A.M.; from that time on he has been comfortable. He had a normal movement this morning and another this afternoon without blood. This afternoon he had very slight pain.

19*th*. Last night a slight eruption was noted. To-day, scattered over the legs, feet, and buttocks, are fine petechiæ, and on the calf of the left leg there is a bluish ecchymosis about 2.5 x 1 cm. in diameter. On both elbows there is an eruption formed by the coalescence of rose-colored, slightly raised papules. He has lost his appetite. The tongue is again lightly coated.

20*th*. The little fellow had an uncomfortable night, being restless and complaining of pain in the abdomen. This morning his appetite is poor. Temperature, 98.8° F. The petechiæ persist, and in addition he has a subcuticular steel-blue ecchymosis over the right hypothenar region. It is here that he complains of pain. The eruption on the right elbow has almost disappeared, leaving little brownish discoloration. On the left it still persists, although less evident. On pressure it is

not entirely effaced, still leaving a brownish color. The urine is yellow and looks a little smoky; acid, 1009; moderate flocculent sediment, containing a few hyaline casts and red blood corpuscles; albumin is present. His diet is now again limited to soft food. Castor oil is administered, and calcium chloride, 10 grains, three times a day is being given.

24*th*. The boy is again comfortable. He is very hungry; he looks pale. The eruption has disappeared, with the exception of a bluish discoloration on the calf of the left leg, representing the old ecchymosis. The colicky pain during the foregoing illness occurred in paroxysms at intervals varying from five minutes to an hour. On the 12th the pain was almost continuous. Between the attacks the little fellow would fall asleep, only to be awakened by pain, which caused him to roll about in great discomfort. So severe was it that he would cry out. The left lateral decubitus and lying on his face were the favorite positions during the painful periods. The abdomen was exquisitely tender during the colic. Throughout the illness the abdomen was slightly distended. The spleen was not palpable. The constant feature was a fine gurgling detected by palpation of the left half of the abdomen, particularly in the upper quadrant. The days were relatively comfortable, with the exception of the 12th, when the pain was almost continuous. The colic appeared toward evening, increased in severity through the night, reaching its maximum between midnight and three o'clock; then a decrease in its intensity and a more comfortable morning.

April 7th. A specimen of the urine sent from Atlantic City, where the patient went March 28th, is pale yellow, with a moderate flocculent, grayish sediment, containing many red corpuscles, some round granular cells, and an occasional hyalogranular cast, 1008; albumin present, 0.5 per cent.

11*th*. The patient returned from Atlantic City, having gained weight and looking very well. Although the complexion is good, the conjunctivæ look a little pale. While in Atlantic City he once suffered from a little pain in his abdomen, and his mother noticed a few "spots" on his legs. The urine of to-night is pale yellow, acid, 1008; small flocculent sediment, containing many red blood corpuscles, a few hyaline casts. Albumin is present.

15*th*. The patient has been in bed and on strict milk diet, in spite of his dislike for this food. To-day he was much nauseated and vomited directly after taking food.

16*th*. The urine is brown and smoky and deposits a reddish-brown sediment containing blood, some round granular cells, and granular casts, 1024; contains much albumin. Very little fluid has been taken during the last twenty-four hours.

From the time he improved the albumin lessened. There were very few tube casts, and at the date of the last report, May 10th, he was doing well, though still kept in bed and on a milk diet as precautionary measures.

A criticism has been made on my previous papers that I had jumbled together a motley group of cases, some of purpura, some of angioneurotic œdema, others of peliosis rheumatica; others, again, of exudative erythema. I did so on purpose, for I was seeking similarities, not

diversities, and I refrained as much as possible from the use of specific terms, often, indeed, not knowing what to call a case watched for a long period. What, for instance, shall we call Case II., which I studied at intervals for six years—purpura, erythema, angioneurotic œdema, or urticaria? The lesions varied from time to time, but there could be no question that all of them, with the extraordinary visceral symptoms, were manifestations of one and the same cause. Or Case XXVI., beginning with simple erythema, then crops of urticaria, and an exudative erythema with purpura and vesiculation? In Cases XIX. and XXVI. the most expert dermatologists were in doubt as to the nature of the lesions, and could only say that it was a peculiar type of erythema. On the other hand, the members of the erythema group have not all the same etiology, and, indeed, as is well known, the individual members have a very diverse etiology. For example, the urticaria of cholelithiasis, of an ague paroxysm, that caused by eating shell-fish or strawberries, the urticaria of hydatids, and an asthma attack has the same clinical and anatomical features, though caused by a variety of poisons —bacterial, protozoal, vegetable, and metabolic. It is not unlikely that the poison in itself, of whatever kind, is of less intrinsic importance than certain transient aspects of cell metabolism. In the first place, there is no constancy of action of the same poison in different persons, or even in the same person at different times. This is notoriously the case with the animal and vegetable substances causing urticaria. In the second place, the chronic forms of urticaria probably illustrate a morbid and persistent sensitiveness of the cutaneous vessels to poisons of either intestinal or tissue origin. And, thirdly, the importance of the local status is shown in that remarkable form of urticaria which comes on after exposure to cold. So long as the face is at a temperature above 60° F. the patient is all right; exposure at 40° F. is followed at once by an outbreak of urticaria. And, lastly, a peculiarity that may be transmitted through several generations, as in angioneurotic œdema, which is only urticaria "writ large," must either be a morbid susceptibility of tissue or an inherited peculiarity of metabolism, or both combined.

The relation of the erythemas to infective processes is interesting. Certain types of exudative erythema behave like an acute febrile disease, and Dühring, of Constantinople, has described an epidemic form. In many of the fevers—typhoid, pneumonia, rheumatic fever, etc.—there may be symptomatic erythemas. The severer forms of purpura urticans with arthritis and colic (Henoch's purpura and the peliosis rheumatica) may run an acute febrile course with heart complications. Many of the graver cases of purpura have followed an acute infection, puerperal fever, gonorrhœa, etc., and in No. V. of my series the patient had recently had gonorrhœa, and in Nos. XXI. and XXIX. there had been

otitis media. The rheumatic poison is probably responsible for very many cases. But in a very large group the condition persists for so many years, as in Cases II., XIV., XV., and XXVII., that an infective process is out of the question.

The visceral lesions are most diverse in situations and form, and vary a good deal with the character of the eruptions. It has to be borne in mind that certain skin lesions are associated secondarily with diseases of the internal organs. Purpura of a severe type is very common in Bright's disease; urticaria and purpura in cirrhosis of the liver and cholelithiasis; urticaria in asthma, and all forms of erythema with the chronic valvular lesions of the heart in children. It may not always be easy to say, particularly in asthma, which is the primary, whether the urticaria has preceded the asthma, or *vice versa*.

The complications form two great groups—the angioneurotic and the inflammatory. To the former belong the swellings of the fauces, the œdema of the glottis, the changes in the bronchial mucosa, causing asthma, and the colic, which is probably due to localized œdema of the gastrointestinal walls; to the latter the more serious complications, endocarditis, pericarditis, pleurisy, pneumonia, and nephritis, but, as it is not easy to make a sharp distinction in all instances, it will be better to discuss the complications according to the organs attacked.

Cerebral. In two cases there were brain symptoms, active delirium in Case I. at the time of recurring attacks, while in Case XV. there were five or six attacks of aphasia and hemiplegia, and the cerebral and cutaneous manifestations lasted for thirteen or fourteen years! It seems not improbable that these transient attacks were due to vascular changes in the brain, the counterpart of those occurring in the skin. An analogous condition is met with in Raynaud's disease, of which I have reported a remarkable instance,[1] with repeated attacks of monoplegia, aphasia, and hemiplegia, alternating or occurring at the same time with the local syncope or asphyxia of the fingers and toes.

Respiratory. Swelling of the fauces and larynx may cause severe dyspnœa and attacks like croup. In the family I described with angioneurotic œdema two members died of œdema of the glottis. In Case XXV. of the series the lad had much swelling of the fauces and croup-like attack, twice accompanied with swelling of the throat and neck, evidently due to œdema.

The association of asthma with urticaria and other skin diseases of the erythema group has long been known, and one of the last papers of our lamented friend Dr. F. A. Packard dealt with this subject.[2] It seems to be most frequent with urticaria. There was no instance in my series, though in Case II. there was a persistent catarrh of the smaller tubes leading to emphysema.

[1] The American Journal of the Medical Sciences, 1891. [2] Ibid., 1896.

Three of the patients had severe pneumonia. In Case II. it occurred toward the close of the remarkable illness of five years' duration. In Case XI. the child died of croupous pneumonia a month after the onset of the third attack of purpura. It was impossible to say whether there was any connection between the two diseases. In Case XXVI. there was a protracted pneumonia following directly upon a severe outbreak of exudative erythema. It is likely that the recurring skin lesions, the pleuropneumonia, the phlebitis, the general glandular enlargement, and the fatal nephritis were due to one and the same poison.

Cardiac. Acute endocarditis seems a rare complication. I saw an instance in Philadelphia, and there are a good many cases in the literature, usually in the intense arthritic purpura (peliosis rheumatica). Only three cases in my series had heart murmurs, and in none of the patients was it likely that endocarditis existed. Pericarditis occurred as a terminal event in Case II.

Gastrointestinal Symptoms. One of the most constant features in this whole group, occurring in twenty-five of the cases, is the recurring attacks of colic, sometimes with vomiting, sometimes with diarrhœa, occasionally with the passage of blood. The association of attacks of colic with outbreaks of urticaria has long been known. Analyzing the series here reported there were eight cases in which the colic occurred alone with the outbreak of the skin lesions; in fifteen there were in addition gastric attacks, nausea and vomiting, and in five diarrhœa, and in eight blood occurred with the colic. The colic is severe, and the attacks may persist for an hour or more and may require morphine for relief. The diagnosis of renal colic was made in one case (II.), and in Case XX. the patient was admitted to the surgical ward for appendicitis. Two interesting associations of the colic may be referred to here. In certain cases of angioneurotic œdema, a malady which belongs to this erythema group, colic is a special feature, and in the family I reported almost all of the cases throughout the five generations had colic with the outbreaks of the œdema. Still more interesting is the relationship which this condition bears to those obscure cases of recurring colic and gastrointestinal crises in children, many of which I believe belong to this group. Take, for example, the following case:

Charles E. D., aged thirteen years, referred to me by Dr. Dick, of Salisbury, Md., on February 8, 1903, complaining of attacks of severe cramps in the abdomen, which have recurred at intervals for nine years. At first they occurred every month or two, and were associated with vomiting of green bile. Now he does not vomit. Of late years they have recurred with much greater frequency, so that now he never passes a week without an attack. So far as I can make out there is nothing in the diet which ever makes any difference. He is better in

warm weather, and more often has the attacks in damp weather, in winter, and after prolonged exertion. He never has any diarrhœa. He has had swelling of the joints, occasionally has had growing pains and pains about the knees. The attacks begin abruptly. Occasionally he has a little premonition in the way of uneasy sensations. The doctor states that he writhes about the floor in the attack and doubles himself into a knot and squirms about the floor like a snake. As the father expresses it, the contortions are awful. Last week he had an attack every day, but not of great severity, and this is the rule. Attacks at long intervals are always more severe. The examination of the child was negative. The urine was negative, except that the morning specimen showed a slight trace of albumin. The child was not very well grown, and looked pale and delicate. The spleen was not enlarged.

Naturally I asked particularly about the occurrence of hives or of any other skin rash, or the occurrence of arthritis, but the history was entirely negative. What is particularly interesting about the case is the fact that his mother had similar attacks during her early life, which lasted until she reached puberty. The boy has three sisters, each one of whom suffered in early life severely with hives, but had no abdominal pains.

Cases XVIII. and XXVII. show the connection of this type of colic in children with the condition under discussion. For years there were attacks of colic of great obscurity. In the former case the child was admitted with a typical outbreak of purpura and erythema; in the latter the skin lesions have lately given a clue to the nature of the protracted abdominal attacks. In angioneurotic œdema the patients may have colic alone without the giant urticaria. In children recurring colic with nausea and vomiting may be sometimes a gastrointestinal counterpart or equivalent of a cutaneous attack, as they certainly are in angioneurotic œdema, and, as I think, we may take it for granted they were in Cases XVIII. and XXVII.

Renal. In fourteen of the cases there was acute nephritis, indicated by albumin and numerous tube casts, and in seven cases blood. In Case VII. there was albumin without any blood or tube casts. In two cases the picture was that of an acute nephritis with dropsy. The seriousness of the nephritis is shown by the fact that of the seven deaths among the twenty-nine patients five died of uræmia. The importance of the poisons causing the skin lesions of the erythema group is not sufficiently recognized as a cause of acute nephritis. Dickinson makes no mention of it in his article in Allbutt's *System*, and in most of the other systematic writers there is a corresponding silence on the subject. The nephritis, as a rule, comes on at the height of the skin lesion, or it may follow within a week or ten days, or even, as in Case XXVI., a couple of months after the subsidence of the skin lesion. There may

be no dropsy even with an intense nephritis (Case II.). The œdema of the skin lesion may simulate the puffiness of the face in renal dropsy. This was very marked in Case XXV. The photograph sent by Dr. Love showed a typical renal facies, but in this boy the urine had never been albuminous. Five of the fourteen cases of nephritis died with uræmic symptoms. With reference to the skin lesions in the renal cases, in four purpura alone was present; in three, purpura and urticaria; in two, purpura, urticaria, and erythema; in one, purpura, erythema, and œdema; in one, at various times purpura, simple erythema, urticaria, and localized œdema were present; in one, œdema and erythema, and in two, purpura and erythema. Purpura was the most constant lesion, occurring in thirteen of the fourteen cases with nephritis. It is interesting to note that there was not an instance of nephritis with œdema alone, and I have never met with it in pure cases of angioneurotic œdema.

The morbid anatomy of this type of nephritis has not been very carefully studied, and I have no post-mortems on any of the fatal cases in my series. Dr. W. T. Watson,[1] of Baltimore, has reported a remarkable case in a girl, aged ten years, who had had for two or three months transient pains in the abdomen. On the evening of January 28th she had a chill, followed by vomiting and fever; on the 31st, patches of erythema and maculæ; on February 1st, arthritis; on February 2d, purpuric spots; on February 3d, acute nephritis. On March 1st there was an aggravation of the condition with blood in the urine. On March 5th there was almost complete anuria, followed by general anasarca and death on the 12th. There was no endocarditis. The spleen was enlarged and firm. The kidneys were immensely enlarged, measuring each 12 x 7 cm. The cortices were pale, the striations distinct, the glomeruli prominent as gray, translucent nodules, almost like miliary tubercles. There were some small hemorrhages. Dr. W. McCallum, who made the autopsy, reports that microscopically the kidney shows extensive degeneration in the renal epithelium. Many tubules are partly filled with desquamated and degenerated epithelial cells, while the lining of others is ragged and shows fatty change. The tubules contain, besides the desquamated cells, many hyaline casts, or are sometimes filled with red blood corpuscles. The glomeruli form the most striking feature in the section. The Malpighian tuft is in almost everyone much compressed by the new growth of a mass of cells in the area of the capsular space which forms a crescentic mass. These cells lie in a connective-tissue network, which is continuous with the connective tissue outside the capsule. They have often small, subdivided, capsular spaces lined by capsular epithelium, suggesting that

[1] Maryland Medical Journal, 1903.

the original capsular space was merely invaded by this new growth. Dr. McCallum tells me that the case belongs to a group which has been lately described under the name of adhesive glomerulonephritis, in which there is not only a proliferation of the epithelial cells, but also a new-growth of connective tissue in the capsules.

Arthritis or arthritic pains were present in seventeen cases. The joint lesions were slight, as a rule; in no case was there severe polyarthritis. The relation of the rheumatic poison to the arthritis and the other lesions is clear enough in some cases, but we cannot say that the arthritis is a hall-mark by which we can always recognize the rheumatic poison. A great many of the cases of arthritic purpura or the peliosis rheumatica have, I believe, nothing to do with the poison of rheumatism. On the other hand, erythema, with or without purpura and arthritis, may be in children, as are endocarditis, tonsillitis, and subcutaneous fibroid nodules, manifestations of the rheumatic poison—links in the rheumatic chain.

Hemorrhages from the Mucous Membranes. There may be recurring severe attacks of epistaxis, as in Case X.; more frequently they are slight. With the colic blood may be passed, usually in small amounts; it occurred in seven cases. Blood was passed with the urine in eight cases, in all in association with nephritis. In Case XIV., after outbreaks of urticaria for years, the final symptoms were those of a severe purpura hemorrhagica.

Lastly, it is of interest to tabulate the cases according to current nomenclature of skin diseases. In five purpura was the only lesion; in four, with arthritis; four, with fever. In seven there was the common combination of purpura with raised wheals—purpura urticans—which is the usual lesion of peliosis rheumatica. In five cases there was angioneurotic œdema, sometimes occurring alone, more frequently with other erythematous lesions, or alternating with them. Erythematous lesions occurred in fourteen cases, in only two alone, with swelling in the form of erythematous blotches, usually with purpura or urticaria, once only with vesiculation of an extensive character.

A word as to treatment. The very chronic cases with recurring colic for years may resist all measures. Alterative courses of gray powder, with careful dieting, may be helpful. With angioneurotic œdema nitroglycerin in full doses may be tried. Dr. Love writes that in his remarkable case (No. XXV.) camphor has done good. In such severe types as Case XIX. nothing seems to arrest the progress. The chief danger is from the kidneys, and in so frequently presenting this subject I have hoped to impress upon my colleagues the importance of recognizing this form of nephritis, and of taking early precautions to prevent its progress, against which I think protracted rest in bed and a milk diet are the best means at our disposal.

List of Cases with Visceral Lesions of the Erythema Group.

No.	Name.	Age and sex.	Skin lesions.				Fever.	Colic.	Vomiting.	Diarrhœa.	Hemorrhages.	Nephritis.	Albumin.	Arthritis or arthritic pains.	Endocarditis.	Enlarged spleen.	Other complication.	Mode of death.	Duration.	Remarks.
			Purpura.	Urticaria.	Œdema.	Erythema.														
1	B. L.	M. 27	...	...	...	1	1	1	1	...	.....	...	...	...	...	1	.....	.....	6 yrs.	Delirium in attacks.
2	W. E. B.	M. 11	1	1	1	1	...	1	...	...	Nose.	...	...	1	...	1	Bronchitis, cough, emphysema, pneumonia.	Pericarditis.	5 yrs.	
3	I. W.	M. 6	1	1	...	...	...	1	...	1	Bowels.	1	1	1	...	...	.....	Dropsy & uræmia.	10 wks.	
4	B. H.	M. 46	1	1	...	...	...	1	1	1	.....	1	1	1						
5	J. D.	M. 38	1	1	...	1	1	1	1	...	Urine.	1	1	1	...	...	Gonorrhœa.	.....	2 mos.	Came on after gonorrhœa.
6	W. L.	M. 9	1	...	...	...	1	1	1	...	Bowels.	1	1	1	...	...	.....	Pneumonia.	Third attack.	Death from pneumonia a month after onset of third attack.
7	W. R.	F. 4	1	1	1	...	1	1	...	...	.....	...	1	1	...	...	.....	.....	About 3 wks.	
8	O. L.	F. 5	1	...	...	...	...	1	...	1	Urine.	1	1	1	...	...	.....	Uræmia.	.....	General anasarca.
9	L. J.	M. 12	1	1	...	...	...	1	1	...	.....	...	...	1	...	...	.....	.....	.....	Remarkable case; symptoms like purpura hemorrhagica.
10	B. W.	M. 30	1	1	...	...	...	1	1	...	Nose, recurring attacks; gums.									
11	A. R.	F. 18	1	1	...	...	...	1	1	...	Stomach, bowels, lungs.	...	...	1	...	...	.....	.....	4 yrs.	Recurring attacks of great severity.
12	B. L.	F. 24	...	1	...	1	1	1												
13	R. B.	F. 49	...	...	...	1 exnd.	1	1	...	...	.....	...	...	1	...	1				
14	A. D.	M. 57	1	1	...	...	...	1	1	...	Stomach, bowels, urine.	1	1	...	...	..	Bronchitis.	Uræmia.	27 yrs.	Extraordinary case; hemorrhages in final attack.

15	C. A. R.	M. 29	1	1	1	...	...	1	...	..	Urine.	1	1	...	...	...	Aphasia and hemiplegia, 5 or 6 attacks.	.....	13 to 14 years.	Remarkable case.
16	H. L.	M. 11	1	...	...	...	...	1	1	...	....	...	...	1						
17	G. K.	M. 18	...	1	...	1	...	1	...	...	....	...	...	1	Apex mur.					
18	B. P.	F. 7	1	...	...	1	1	1	1	...	Bowels.	...	...	1	...	1	.....	.....	.....	Remarkable case; recurring colic for five years; case like No. 26.
19	E. C.	F. 15	...	...	1	1	1	...	...	...	.....	1	1	...	...	1	Otitis, enl. lymph glands.	Uræmia.	7 mos.	
20	M. R.	M. 15	1	1	...	1	...	1	...	...	Bowels. urine.	1	1	...	...	1	.....	.....	.....	Admitted for appendicitis.
21	J. D.	M. 12	1	...	...	...	1	1	1	...	Urine.	1	1	...	...	...	Otitis.			
22	E. C.	F. 6	1	1	...	...	...	1	...	...	Urine.	1	1	1						
23	S. E. W.	F. 14	1	1	...	1	1	1	1	1	.....	..	...	1	1 mur.	1				
24	M. S.	M. 3	1	...	...	1	...	..	...	...	.....	1	1							
25	N. D.	M. 14	...	1	1	1	...	...	...	...	.....	...	...	...	...	...	Swelling of throat, edema of face, spasmodic croup.	.....	.....	Remarkable case.
26	L. E.	F. 24	1	...	...	1	1	...	...	...	.....	1	1	...	...	...	Pneumonia, thrombosis left femoral vein, enlargement lymphatic glands.	Uræmia.	8 mos.	Extraordinary case, like No. 19.
27	D. B.	F. 13	...	1	...	1	1	1	1	...	.....	...	...	...	Syst. mur.	...	.....	.....	.....	Colic from infancy.
28	A. S. A.	M. 26	1	...	...	...	1	1	1	1	Bowels.	...	...	1						
29	E. F.	M. 5	1	...	...	...	1	1	1	...	Bowels.	1	1	1	..	...	Otitis media.			

A CLINICAL AND PATHOLOGICAL STUDY OF TWO CASES OF SPLENIC ANÆMIA, WITH EARLY AND LATE STAGES OF CIRRHOSIS.

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INTRODUCTION.

There is a growing belief in the existence of a disease described under many names—always a sign of ignorance—but at the present time called splenic anæmia more frequently than by any of the other synonyms. The term has been loosely applied, and, in fact, invites loose application, unless one has some acquaintance with the literature of splenic diseases and remembers "the good rule laid down by Socrates," as quoted by Osler. With that precaution, we think with Osler and Senator that splenic anæmia is a better word for the purpose than any other yet suggested. Since Banti showed the evolution of certain cases with enlarged spleen and anæmia into later stages with ascites and cirrhosis of the liver, his name has been used by some as a synonym, and, although Osler's suggestion that it be applied only to the later stages will doubtless have many followers, it will often be difficult to draw a line; for, though Banti thought he had discovered a disease distinct from splenic anæmia, he was aware of the difficulty of making a final conclusion. Even in the more recent literature, under the names splenic anæmia and Banti's disease, we find, besides some obviously faulty diagnoses, certain cases that differ from the generally accepted picture in some details. Senator, in adding greatly to our knowledge, showed that the symptomatology of the blood is not as Banti described it. Since not only this, but also many other allied topics in splenic pathology are only beginning to receive investigation, it is necessary to enlarge the casuistry and to collect and report as many cases as possible in which the clinical and anatomical features have been carefully studied. Banti's suggestion of extirpation of the spleen has already been of great benefit, not only as a therapeutic measure, but by increasing our knowledge of the changes in different stages, and should be followed in well-selected cases.

In the following we report two cases bearing upon the subject. One of these seems a good example of splenic anæmia in a compara-

tively early period. Clinically it is a good example of splenic anæmia in the old sense, but anatomically it shows changes in the liver that indicate the occurrence of cirrhosis of the liver had the condition gone on longer. The other has many of the features of Banti's late stage, but is complicated by an old and obscure peritonitis. Since this does not seem to have causal relations with the important changes in the liver and spleen, we report the case on account of the other changes. We do not intend to consider now the literature of the subject, reserving that for another time.

Part I. Clinical (Dr. Dock).

Case I. *Splenomegaly, anæmia; removal of spleen; death. Fibrosis of spleen; beginning fibrosis of liver; stenosis and calcification of the portal vein; hyperplasia of hæmolymph nodes; new formation of lymphoid tissue.*—I. T. A., aged forty-one years, was admitted to the University Hospital as an out-patient July 27, 1899.

Family History. Father died at seventy-two years, having had a cough for twenty years. Mother died at seventy-eight years, of pneumonia. Patient had six sisters and three brothers; two brothers and two sisters are alive. Two sisters died of consumption, one in childbirth, one of "stomach disease." One brother died of "brain disease." Patient has one daughter, aged ten years, in good health. (Though a strong and robust girl, she had extensive pharyngeal adenoids, besides enlarged tonsils, and some months after this underwent operation for appendicitis, when the lymphoid tissue of the appendix was found to show a marked hyperplasia without other signs of appendicitis.) The patient's wife died one year ago, of leukæmia. (She was treated as an in-patient and out-patient of the Medical Clinic for a year and a half, during which time the husband showed a great interest in splenic disease, bringing to the hospital several patients supposed to have enlarged spleens.)

Habits and Occupations. Patient lived on a farm in his youth; since the age of twenty-one years has been in mercantile business. Has worked hard and at long hours, but has not suffered from that, and has not been unusually exposed. Meals have been regular; food good. Appetite has always been good, but bowels irregular. Has used little tea and coffee for ten years; alcohol rarely; tobacco not at all.

Previous Diseases. Had the usual diseases of children; had a severe fever, probably typhoid, seventeen years ago, and a mild attack of influenza a few years ago. Had always been subject to sick headache, but in 1901 had rectal "polyps and ulcers" operated (by a notorious "orificial surgeon"), and headaches have been less severe since then. Most careful inquiry could elicit no history of venereal disease.

Present Disease. About a week ago first noticed a soreness in the left hypochondrium and uneasiness in the abdomen, and on feeling discovered a tumor in the left side. He had noticed a gradual enlargement of the abdomen for a year, but thought nothing of it.

Status præsens, July 27, 1899 (Dr. Arneill). Frame of medium size, rather slender; muscles small and soft; panniculus thin; weight, 126 pounds, the usual weight in summer. The bones and joints are normal;

the inguinal glands can be felt; the other superficial lymphatic glands are not enlarged.

The gait is normal, appearance somewhat cachectic, expression depressed, mind clear.

The skin is somewhat pale and sallow, the visible mucous membranes of fair color.

The teeth are artificial; the tongue is of medium size; has a thin, white, moist coat.

The thorax is of good size and shape; expansion moderate; percussion and auscultation of lungs negative.

The apex-beat is in the fourth interspace, one inch inside the nipple line; the dulness is not enlarged; the sounds normal; no murmurs.

The abdomen is an inch above the level of the ribs, larger in the left side. The liver dulness extends from the sixth interspace to the edge of the ribs; the thin edge can be felt an inch below the ribs in deep inspiration. The splenic dulness begins on the seventh rib, in the mid-axillary line; extends through Traube's space to the navel line in the left nipple line, and two inches below the navel in the middle line. On palpation a hard mass can be felt in this region, having a sharp angle two and a half inches below the navel. A sharp edge runs up one inch to the right of the navel, and shows a notch an inch above the navel.

The radial artery is somewhat tortuous, the pulse easily compressed, moderately full, tension medium, rate 90, regular. The veins of the thorax, especially on the right side and shoulder, are easily seen. The patient says this has always been so.

The urine shows a trace of albumin; no casts; otherwise negative.

The blood: red corpuscles, 4,480,000; leukocytes, 5857; hæmoglobin, 75.

July 29th. Test breakfast shows good digestion and motor power; Congo positive; Günzberg negative. The patient was ordered Fowler's solution and dilute hydrochloric acid.

August 29th. Patient returns for examination, saying he feels better at times. He complains of pain in the right side. The tumor is still present, the boundaries being the same as in the previous examination. Leukocytes, 6366.

September 20th. The patient is still sallow and cachectic, the spleen slightly smaller than at the last examination.

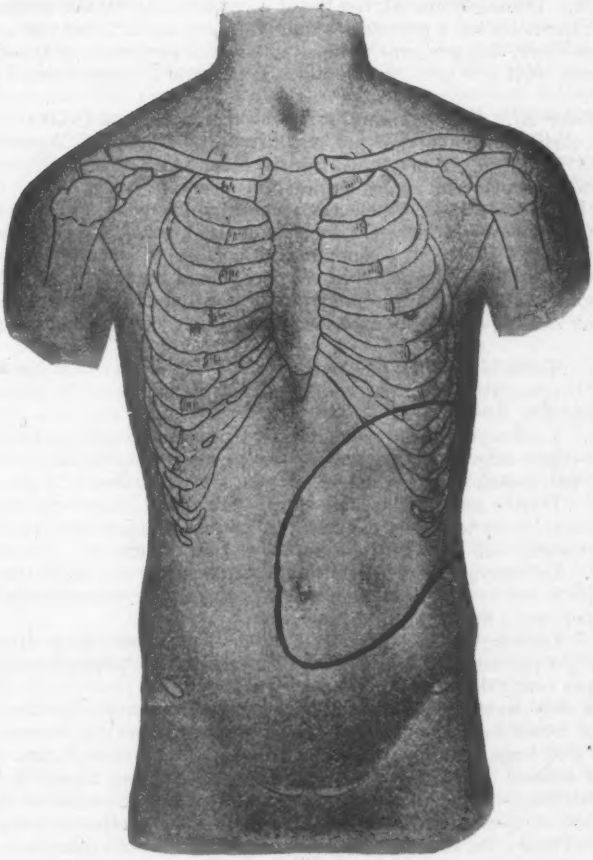
Examination of the stained blood taken at the early visits had shown red corpuscles of fairly uniform size, a few normoblasts, no megaloblasts or myelocytes. Returning from my vacation, I examined, with great interest, the specimens that had been preserved. Finding nothing essentially different from what is noted above, I sent for the patient, found the history and physical condition accurately described, and made further examinations of the blood.

22*d*. Differential count (Dr. Dock). Small lymphocytes, 32 per cent.; large lymphocytes, 5 per cent.; large mononuclear and transitional, 6 per cent.; polymorphonuclear, 52 per cent.; eosinophiles, 4 per cent.; degenerates, 1 per cent.; normoblasts, 4 to 100 leukocytes. The red corpuscles vary considerably in size and shape, but there are no very large or very small cells. Many cells show vacuoles. There are no polychromatophiles. The normoblasts have the usual characters well marked. Only one shows irregular staining of the nucleus. The polymorphonuclear leukocytes show a good many large ones, with small,

deeply-staining nuclei. The lymphocytes are mostly very small, the nuclei staining deeply and showing fragmentation. The eosinophile cells are all of normal appearance.

The general appearance of the blood resembled that often seen in pernicious anæmia at times of comparatively good health. Numerous

FIG. 1.



CASE I.

examinations showed a total absence of megaloblasts and of Ehrlich's myelocytes, as well as of any other indication of bone-marrow disease. The diagnosis of leukæmia in the aleukæmic stage, such as the patient's wife had at one time, was ruled out, and as the result of a differential diagnosis not necessary to detail here, splenic anæmia was accepted. In view of the theory of Banti and of the operations based on it or a

similar theory, I advised an attempt at removal of the spleen, which, after some consideration, the patient accepted. The advice was given on October 2d, but as the patient wished to arrange his affairs before the operation, he was examined at long intervals in the next month. In this period there was an interesting change in the proportion of the various forms of leukocytes, as appears best from the following counts, which include all up to the last day:

29*th*. Hæmoglobin, 60; red blood corpuscles, 3,640,000; leukocytes, 2037; normoblasts, 1 per cent.; small lymphocytes, 22.5 per cent.; large lymphocytes, 3.6 per cent.; transitionals, 1.5 per cent.; polymorphonuclears, 60.1 per cent.; eosinophiles, 5 per cent.; degenerates, 7.3 per cent.

October 2*d*. Hæmoglobin, 70; red blood corpuscles, 4,288,000; leukocytes, 2546; normoblasts, 1 per cent.; small lymphocytes, 25 per cent.; large lymphocytes, 3 per cent.; transitionals, 3 per cent.; polymorphonuclears, 63 per cent.; eosinophiles, 3 per cent.

15*th*. Normoblasts, 1 per cent.; small lymphocytes, 15.5 per cent.; large lymphocytes, 5 per cent.; transitionals, 1 per cent.; polymorphonuclears, 75.5 per cent.; eosinophiles, 3 per cent.

November 1*st*. Hæmoglobin, 75; red blood corpuscles, 4,300,000; normoblasts, 0.9 per cent.; small lymphocytes, 18 per cent.; transitionals, 7.5 per cent.; polymorphonuclears, 78 per cent.; eosinophiles, 3.5 per cent.

3*d*. Normoblasts, 1.1 per cent.; small lymphocytes, 8.6 per cent.; large lymphocytes, 13.2 per cent.; polymorphonuclears, 76 per cent.; eosinophiles, 2.6 per cent.

4*th*. Leukocytes, 3056; normoblasts, 1 per cent.; small lymphocytes, 6 per cent.; large lymphocytes, 4.33 per cent.; transitionals, 2.66 per cent.; polymorphonuclears, 85.66 per cent.; eosinophiles, 1.33 per cent.

6*th*. During anæsthesia, leukocytes, 4909; small lymphocytes, 6 per cent.; large lymphocytes, 4 per cent.; transitionals, 4 per cent.; polymorphonuclears, 84.5 per cent.; eosinophiles, 1.5 per cent.

7*th*. Leukocytes, 18,080; normoblasts, 4 per cent.; large lymphocytes, 1.5 per cent.; transitionals, 4.6 per cent.; polymorphonuclears, 91.9 per cent.; degenerates, 1.9 per cent.

9*th*. Leukocytes, 21,000; normoblasts, 6 per cent.; large lymphocytes, 2.9 per cent.; transitionals, 4.6 per cent.; polymorphonuclears, 90.8 per cent.; degenerates, 1.4 per cent.

Not only were the small lymphocytes less numerous, but the most typical forms were even fewer than the figures indicated, because the small and large lymphocytes showed many intermediate forms. The nuclei stained less intensely, but clearly, resembling those of large lymphocytes. Degenerated leukocytes were usually numerous up to the time of operating, and were nearly all of the polymorphonuclear class. These cells, therefore, were more numerous than in the beginning of the observation, but showed an unusual tendency to break down. While they are largely artefacts as found in the preparations, the variable numbers are suggestive.

October 31*st*. Patient enters hospital. Appetite is good; general condition as before.

November 3*d*. The spleen extends from one and a half to two inches below the navel, in the middle and left parasternal lines. There is a palpable groove half an inch above the navel.

6*th*. The spleen was removed under ether anæsthesia by Dr. C. B. Nancrede. There was considerable distention of the intestines. The spleen had very few adhesions at the anterior surface, but there were many at the upper end. The upper end was unusually thick in proportion to the other dimensions, and this, with the adhesions and the great size of the vessels, made the removal unusually difficult. Slight symptoms of shock appeared as the vessels were being tied. The pulse and respiration were good all through, but perspiration became profuse just at the end. After the operation there was nausea, tympanites, frequent and small pulse; the mind was clear. The patient, who had before looked on the operation with calmness, became very anxious, and on November 9th, three days after the operation, became collapsed, with cold skin, sweating, pallor, small and soft pulse of 88 beats to the minute, and died on November 10th, at 3.30 P.M.

CASE II. *Intestinal hemorrhage with diarrhœa; ascites; splenomegaly; fibroid tumors of uterus; laparotomy; removal of fibroids; discovery of cirrhosis of liver at operation; death from hemorrhage from varicose vein in stomach; sclerosis and calcification of portal vein; fibrosis of spleen and liver (atrophic cirrhosis); hyperplasia of hæmolymph nodes.*—Mrs. M. S., aged fifty-three years, housewife, born in Michigan, was admitted to the Medical Clinic of the University Hospital October 7, 1902. She had entered the Surgical Clinic for treatment of hernia, but was referred to the Internal Clinic on account of ascites.

Family History. Mother died at age of fifty-eight years, of "consumption." Father died at seventy-two of "heart disease." Four brothers and four sisters are in good health. Habits and mode of life have been good.

Previous History. Patient had whooping-cough, measles, and mumps in childhood, with good recoveries. Menstruation began at fourteen, stopped for a year, and was regular after that, lasting about three to four days, without unusual symptoms. At thirteen had an attack of "dysentery," in which she passed much blood. At nineteen was married. Has never been pregnant. (No reason for this could be discovered. Syphilis seems positively excluded.) Menopause in fiftieth year.

Six years ago the patient had "dysentery," and was confined to bed for six weeks. She passed large amounts of blood with loose and watery stools. Since then she has had similar attacks at intervals. A week before admission had a slight diarrhœa without blood in the stools. After the attack six years ago the abdomen gradually became larger, and at that time Dr. H. A. Shurtleff, of Partello, Mich., discovered free fluid in the abdomen, by physical signs. Soon after that œdema of the legs and ankles came on, lasting for two months. It has recurred at intervals since then, but less severely. The umbilical hernia appeared after vomiting, three years ago, and has grown larger; the inguinal hernia came on after coughing, one and a half years ago.

The patient now complains of constant soreness over the whole abdomen. She is constipated and has frequent desire to defecate, but bowels are moved with difficulty. Appetite fair, but patient eats sparingly on account of feeling of distention and sensation of weight in abdomen.

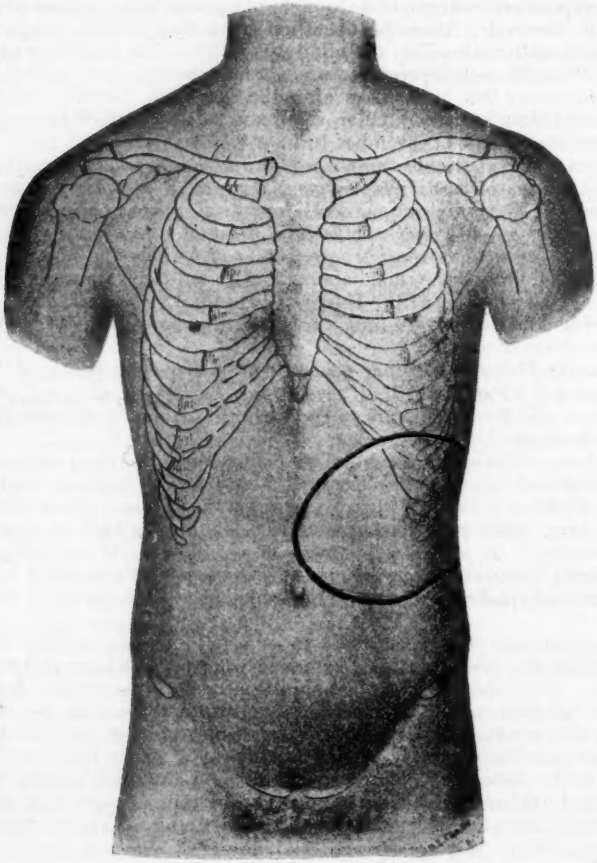
Status præsens, October 7, 1902. Medium height, slender frame; muscles small and soft; panniculus scanty; weighs 120 pounds; best

weight, ten years ago, 150. The joints are normal, reflexes not altered, superficial lymphatic glands not enlarged.

The patient looks weak and cachectic, the expression is weary; mind clear.

The skin is pale, sallow, and inelastic; the visible mucous membrane pale; there is no œdema.

FIG. 2.



CASE II.

The teeth are artificial; the tongue is white-coated.

The thorax is broad, short, wide; epigastric angle wide and lower aperture of thorax distended. The expansion is fairly good, and symmetrical. The right lower lung border is on the sixth rib, the descent limited. Percussion of lung, front and back, negative. Auscultation reveals good vesicular murmur.

Heart. The apex-beat is indistinct, but can be felt in the fourth interspace, just inside the nipple line. There is a faint pulsation in the third left interspace. Cardiac dulness begins on the third rib, extends to within half an inch of the nipple line. Dulness under the sternum from the fourth rib down to the right of the sternum in the fifth interspace, one finger's breadth. The apex sounds are clear, the first accentuated. In the pulmonary area there is a short systolic murmur, followed by an accentuated second sound, lower than the aortic second. Radial pulse slightly tortuous, perhaps slightly thickened; rhythm regular, tension low.

The abdomen is enlarged, measuring thirty-seven inches at the umbilicus. It is bulging at the sides and flattened on top, with a prominence in the umbilical region. (During the examination this prominence subsided, leaving a slight depression in the same place.) The superficial abdominal veins are not enlarged. There are a few large venules over the anterior spines of the iliac bones. In the left side, above and below the iliac crest, some small veins are visible through the skin. On sitting up the protrusion reappears in the umbilical region, becoming larger than before. There is also a wide separation of the recti, permitting a great protrusion all the length of the abdomen. A large hernia also appears in the right inguinal region. The intestines can be plainly felt through the skin in the middle part.

Lying down, there is dulness on percussion in both flanks, almost to the nipple lines, and in front two inches above the pubes. The lines change quickly on change of position. Fluctuation is not distinct. Palpation of the lower abdomen is negative.

The liver dulness reaches to the margin of the ribs in the nipple line. Palpation is unsatisfactory on account of the distention.

Splenic dulness begins at the seventh rib, and is continuous with the dulness in the flank. The lower end can be felt about three inches below the edge of the rib.

Examination of the anus and rectum shows the small atrophic tags of old external hemorrhoids, and very small internal hemorrhoids, without evidence of hemorrhage. The mucosa shows a bleeding spot the size of a pinhead about six inches from the sphincter.

Examination of the pelvic organs (Dr. Peterson): negative.

Examination of urine: negative.

The blood flows freely; is thin; bright red. Red blood corpuscles, 2,800,000; leukocytes, 5857; hæmoglobin, 60 per cent. The differential count gave: small lymphocytes, 6 per cent.; large lymphocytes, 5.9 per cent.; large mononuclears and transitionals, 4.2 per cent.; polymorphonuclears, 81.7 per cent.; eosinophiles, 1.2 per cent.; degenerates, 0.6 per cent.; normoblasts, 3 per mille. The red corpuscles show very slight variation in size and shape.

October 21st. The superficial veins of the abdomen are slightly distended. Abdomen tapped in Monro's point and 3800 c.c. of fluid withdrawn. The fluid is clear, reddish-yellow; specific gravity, 1012. It contains a slight increase of red blood corpuscles. Differential count of the other cells: small lymphocytes, 53 per cent.; large lymphocytes, 8 per cent.; polymorphonuclears, 6 per cent.; large mononuclears, 33 per cent. The large mononuclear cells have the appearance of young endothelial cells; the lymphocytes partly that of endothelial cells, partly that of ordinary lymphocytes.

After tapping, the abdomen measures thirty-two and three-quarter inches at the umbilicus. The spleen can be easily felt three fingers' breadth below the ribs, and reaching to the median line. Liver dulness not quite to edge of ribs. Liver cannot be felt.

22*d*. Patient has been more comfortable since the tapping. There is gaseous distention in the right side. The spleen reaches the navel line in the nipple line and the middle line, four fingers' breadth above the umbilicus. The surface of the spleen is smooth, flat; the edge thick; there is a notch above the lower end, on the inner side.

24*th*. The abdomen is larger; thirty-four and a half inches. Signs of free fluid not so extensive as before the tapping. The ankles are slightly œdematous. The superficial abdominal veins are not enlarged. No murmur in the epigastric region.

Blood. Red corpuscles, 4,266,666; leukocytes, 7894; hæmoglobin, 70 per cent. Differential count: small lymphocytes, 3.4 per cent.; large lymphocytes, 6.4 per cent.; transitionals, 11.4 per cent.; polymorphonuclears, 77 per cent.; eosinophiles, 1 per cent.; degenerates, 0.8 per cent. No nucleated red cells found; a few poikilocytes.

30*th*. The patient has complained much of pain and soreness in the pelvis, which could not be explained by any of the manipulations practised upon her. Another consultation was held with Dr. Peterson, and an examination made under ether. A small, hard mass was found in front of the uterus, apparently a fibroid tumor.

Up to this time the diagnosis had not been sufficiently clear to offer a surgical operation to the patient, who was convinced that medicines would not relieve her. Cirrhosis of the liver, most likely at first glance, was made doubtful on account of the history, the long duration of ascites without more venous enlargement, and the relatively large spleen. A Talma operation did not seem warranted. A late stage of Banti's disease seemed ruled out also by the long duration of the ascites and relatively good condition of the blood. On the other hand, I presented the patient both in clinic and before the Medical Society as a possible case of late Banti, with a complication, perhaps in the pelvis, causing chronic peritonitis, and with the blood picture (polymorphonuclears increased relatively) obscured by the local conditions in the abdomen. Here, too, surgical intervention did not seem more promising than it did on the theory of cirrhosis, because if the case were one of Banti's it was probably past the aid of surgery; and my surgical colleague declined to explore in order to determine that point. Tuberculosis and malignant disease of the peritoneum were ruled out by the long duration and the character of the fluid, as well as other features not necessary to point out. Chronic peritonitis due to an undiscovered disease in the pelvis was a diagnosis I took up several times to explain the ascites as an associated phenomenon with the enlarged spleen, but up to the discovery of the fibroid this diagnosis did not lead to an exploration.

Although the fibroid could not be correlated with the enlarged spleen, or even the ascites, save as an exceptional condition, it gave us something to operate for, and so meet the pressing wishes of the patient as well as our own desires for an exploration. Accordingly, Dr. Peterson operated November 7th. A large amount of fluid having the same character as that previously examined escaped from the incision. An irregular, roundish tumor, one and a half inches in diameter, was found adherent to the lower and anterior part of the uterus. It proved to be

a calcified fibromyoma. The uterus was very small, the fundus round and of stony hardness. It was found to be merely a shell around another calcified fibroid, and was removed. There was no distinct disease of the tubes and ovaries, but there were many adhesions around all the pelvic organs. The omentum was fastened low down by adhesions. The spleen was large, the lower surface uneven. The liver was also uneven to the touch, though as seen by light thrown into the abdominal cavity it showed only slight granular change. There were numerous adhesions around the liver and spleen.

The wound healed well at first, but left a small sinus. The ascites gradually returned, and on December 2 five litres of fluid were removed. Specific gravity, 1012.

December 6th. The patient returned to the medical ward. *Status præsens:* The patient looks tired and sleepy, and complains of feeling sick all over. She has had diarrhœa, with great pain. The stools were dark and watery, and contained fresh blood. The abdomen measures thirty-four inches. The abdominal veins are distinct, but not swollen. Palpation of the abdomen causes pain. The wall is soft in general. There is a flattened mass just above the navel line and two inches to the left of the navel, apparently adherent to the deep tissues. The spleen extends not quite two fingers' breadth below the ribs in the nipple line, three fingers' breadth in the parasternal line. There is a soft, blowing, systolic murmur over the base of the heart. A few crackling râles can be heard in the right axilla.

There had been leukocytosis ever since the operation, from 10,000 to 18,000 per cm., with increased percentage of polymorphonuclear cells and many degenerates.

Red blood corpuscles, 3,040,000; leukocytes, 17,200; hæmoglobin, 40 per cent. Differential count: small lymphocytes, 12.2 per cent.; large lymphocytes, 2.2 per cent.; transitionals, 1.1 per cent.; polymorphonuclear, 84 per cent.; eosinophiles, 0.45 per cent. No nucleated reds. Moderate poikilocytosis.

At 1.45 P.M. the patient vomited half a pint of blood, and twenty minutes later a similar quantity. The blood is bright red, alkaline; contains large clots. At 6.30 P.M. patient vomited about six ounces of bloody fluid. Pulse small and irregular. Patient feels weak, dizzy, and nauseated.

7th. Patient feels better. Has not vomited, but passed bloody fluid per rectum.

8th. Patient is weaker and in great pain. Retention of urine.

9th. Still passing bloody fluid per rectum. Cannot swallow.

10th. Incontinence of rectum.

11th. Died at 12.05 P.M.

Part II. Pathology (Dr. Warthin).

Case I.—Mr. I. A., aged forty-one years, American. Died November 10, 1899, at 3.30 P.M. Autopsy by Dr. Warthin, 7.30 P.M., November 10, 1899.

Autopsy Protocol. Body of average height and build. No anomalies or deformities. No signs of trauma. In the left hypochondrium there is a recent laparotomy wound, extending from the edge of the ribs to within three fingers' breadth above the iliac crest. It is covered with

surgical dressings. On removing these the edges of the wound are found to be separated and a fold of intestine protruding. Edges of the wound are discolored (gray to black), but present no evidences of pus. Abdomen distended above the level of the ribs.

The skin is pale, sallow; there is slight hypostasis posteriorly; small boil on right forearm; teeth artificial; hair is negative. The panniculus is greatly decreased; no œdema; muscles are small and emaciated; rigor mortis is present throughout. The extremities are cold. There are no signs of decomposition.

Brain and *spinal cord* not examined. (No permission.)

Diaphragm at the fourth rib on the right; fifth rib on the left.

Position of thoracic organs is normal. Apex of heart in fourth intercostal space, just inside of the nipple line. The mediastinum is rich in fat; no enlarged glands. The thymus fat is in large amount, and in it there is found a pinkish lobulated body 3 cm. long by 1 cm. in diameter. There is no fluid in the pleural cavity. Both pleuræ are free; there are no adhesions.

The *pericardium* is lax; contains a very small amount of clear fluid. Subepicardial fat is slightly increased.

Heart weighs 320 grams; is about the size of cadaver's right fist. The auricles contain ante-mortem white clots; the ventricles are filled with fluid blood and currant-jelly clot. The heart muscle is firm, brownish-red; papillary muscles are prominent. The left ventricle wall measures 15 to 20 mm. in thickness. The endocardium is slightly thickened. The pulmonary, tricuspid, and aortic orifices and valves are normal. The mitral orifice is rather small; does not admit two fingers. Mitral flaps are slightly thickened and shortened. Coronary vessels are negative.

Right lung weighs 272 grams; *left lung* weighs 336 grams. Both lungs are partly collapsed. Pleuræ negative. Slight anthracosis. On section the parenchyma of lungs is grayish-pink; lower lobes slightly hypostatic; no airless areas; very slight exudate on pressure; bronchi are empty; mucosa pale; pulmonary vessels negative; bronchial glands moderately pigmented; on section negative. A number of enlarged glands along the thoracic aorta. On section they are red and spleen-like. The great vessels of the thorax and the thoracic duct are negative.

Mouth and *neck organs* were not examined.

Abdomen. The omentum is rolled up, greatly thickened, and moderately injected, and is adherent to the diaphragm and anterior abdominal wall in the splenic region, and to the peritoneal surface of the ileum by fresh fibrinous exudate. In the region of the spleen there is a large mass of fresh fibrinous adhesions, but no evidence of hemorrhage. Spleen (see below) is absent, having been removed at operation. Ligatures on stumps of splenic vessels are in position. Splenic vessel filled with fresh red thrombi.

The small intestine is greatly distended throughout. The serosa is cloudy, moderately injected, and everywhere covered with a fine fibrinous deposit. Many of the coils are adherent by coarser strings of fibrin, the adhesions being easily separated. The lower sixteen inches of the ileum lie in the left side of the abdomen, a portion of the ileum projecting through the laparotomy wound. Coils of the ileum are adherent to each other by fibrinous exudate, the adhesions being firmer in neighborhood of wound. They are also adherent to the large intestine

by a similar fresh fibrinous exudate. About two and a half inches above the cæcum there are several (six) dark patches in the intestinal serosa, covered with greenish-gray fibrin. These patches are irregular, not triangular in shape, and do not extend entirely around the bowel; some reach about two-thirds of the way. From the serosa these patches extend some little distance into the fat of the mesentery, which shows a superficial necrosis corresponding to the patches of fibrin. These areas have the appearance of having been cauterized; they are all on the upper surface of the mesentery.

The *stomach* is greatly distended, its wall very thin. It lies pressed against the anterior abdominal wall, and was cut during the median incision, letting out a large quantity of sour-smelling gas under high pressure. It contains about 500 c.c. of brown, bile-stained, sour fluid. The mucosa of the stomach shows slight post-mortem change. Veins of stomach wall are greatly distended. The large venous branches in the greater curvature contain fresh thrombi. Around the gastro-epiploica sinistra there is a ligature which includes the serosa of the stomach wall, but not the muscle coats. The coronary veins are greatly dilated, as are also the anastomoses with the œsophageal veins.

The *duodenum* is greatly distended with gas; it contains a small amount of grayish, slimy material. Its mucosa is injected and somewhat swollen. Bile passages are patent.

The *ascending* and *transverse colon* and *cæcum* are greatly distended as far as the splenic flexure, the descending colon being more moderately distended. They contain a small amount of formed feces and a large amount of grayish mucus. The sigmoid flexure and the descending colon are unusually pale and nearly white. The *appendix* is long and narrow, extends into the pelvis; no adhesions about it.

Liver weighs 1280 grams, measures 23 x 15½ x 8 cm.; is small; left lobe very atrophic; right lobe shows posteriorly a number of small anomalous lobules. Capsule is thickened, especially near the ligaments. Veins of capsule congested. Small cavernous angioma, size of a pea, on the upper surface of the right lobe. On section the blood-content is found to be fairly rich. In one of the larger branches of the portal there is a fresh but firm red thrombus. The cut surface of the organ is granular, in some areas markedly so; the granules slightly elevated. The largest granules are paler in color than the remainder of the liver parenchyma, which is of a pale-brown color. Outlines of the lobules are distinct over the greater part of the cut surface; they are smaller than normal. The peripheral zone of the lobules is more grayish, cloudy, and apparently swollen. The connective tissue is apparently increased. In some areas the tissue of Glisson's capsule is thickened, hyaline, and scar-like. In about the middle of the right lobe there is an area about 5 cm. in diameter, which is firmer and lighter in color than the other portions of the cut surface, and is slightly elevated and more distinctly granular. In this area the connective tissue seems to be increased to a greater extent, and the lobules are larger than elsewhere.

The *gall-bladder* is distended, containing about 200 c.c. of dark-brown fluid and three grayish cholesterin stones of small size. Its mucosa is very thin, the wall much stretched.

The *portal vein* is extremely distended, and is filled with a fresh but firm mixed clot. The lumen of the vein at its beginning, just above the union of the splenic and superior mesenteric veins, is irregularly narrowed

and partly bridged off by fibrous, valve-like constrictions, which contain calcareous plates. The vein is divided by these fibrous bands into several sac-like spaces, the walls of all these showing calcification. The tortuous lumen barely admits the tip of a lead-pencil or large probe. The stenosis involves the mouth of the splenic vein, almost completely occluding it. The splenic and superior mesenteric veins back of the stenosis are enormously distended and filled with a soft red thrombus, which is continuous with the firmer mixed thrombus filling the portal. In the media of the portal vein at the point of constriction there are thin plates of calcification. The walls of the splenic and mesentery veins are greatly thickened, and present scattered patches of hyalin.

The *pancreas* is of normal size; on section apparently normal, save for congestion and small areas of hemorrhage. The splenic vein and its pancreatic branches are greatly distended, and are filled with firm red thrombi. In the neighborhood of the pancreas there are several glands resembling accessory spleens. They possess very thick capsules.

In the root of the mesentery, particularly near the portion of ileum showing the dark fibrin patches, the lymph glands are slightly enlarged and hyperæmic. Hyperæmic patches are scattered over the surface of the mesentery, in some cases grayish-black; these are covered with fibrinous exudate.

The *retroperitoneal lymph glands* are not enlarged, but are red, firm, and spleen-like. Abdominal aorta, iliacs, etc., negative.

Ureters negative.

Bladder empty, collapsed; mucosa pale.

Adrenals show post-mortem softening of medullary portion.

Kidneys. The fatty capsule of both kidneys is moderately rich in fat; the fibrous capsule strips easily and is not thickened. The right kidney measures 9½ x 5 x 3½ cm., and weighs 128 grams. The left kidney measures 10½ x 4½ x 3 cm., and weighs 112 grams. The surfaces of both kidneys are bright red, and the venæ stellatæ well marked. On section both kidneys are bright red. Cortex of each measures ½ to ¾ cm. Outlines between labyrinths and medullary rays not well defined; glomeruli not visible; consistence firm; bloodvessels engorged; pelvis of left kidney somewhat dilated, that of right negative.

Ureters and *bladder* negative.

Genitals. External genitals negative. Testicles and epididymis negative. Prostate and seminal vesicles not examined.

Aorta, iliacs, iliac veins, femorals, and *vena cava* negative.

Bones. Negative. Right tibia, fourth lumbar vertebra, and several ribs removed. Tibia split longitudinally. Slight hyperplasia of red marrow. Small lymphoid areas throughout the fatty marrow; the largest of these, in the middle of the tibia, measures ½ x ¼ cm.

Peripheral Glands, Nerves, and Vessels. Negative. (Material was taken from all parts and organs of the body, fixed in Flemming's solution, Müller's, mercuric chloride, alcohol and formalin, and embedded in celloidin and paraffin, and stained by ordinary and special staining methods.)

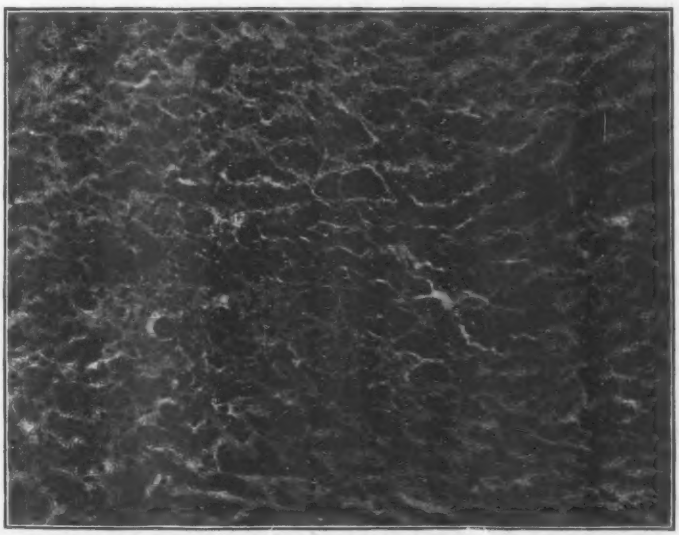
Microscopic. Heart. Sections show atrophy of heart muscle and slight increase of connective tissue.

Lungs. Sections taken from different portions show a number of small recent and old hemorrhagic infarcts. Many of the alveolar walls are thickened, and there is an increase of connective tissue about the

larger vessels. In the lower lobes there is slight emphysema. The bloodvessels are congested, and many of the smaller arteries show sclerotic changes. A few organizing thrombi are found. Anthracosis is moderate. There is a slight œdema. In the capillaries there are found numerous emboli of bone-marrow giant cells, similar to those found in the spleen and liver. These are so numerous that three to six may be found in one field. In the small arterioles the protoplasm of the giant cell is preserved, in the capillaries only the deeply-staining knobbed and lobulated nuclei are visible. A few multinuclear giant cells are found in the arterioles.

Liver. In the sections taken from all parts of the liver, with the exception of the elevated area in the middle of the right lobe, the lobules

FIG. 3.



Liver of Case I. Intralobular increase of connective tissue. Early stage of diffuse interstitial hepatitis. Hyperplasia of intralobular reticulum. × 175.

are atrophic, the atrophy involving chiefly the central portions of the lobules. The central veins and capillaries are dilated, while the liver cells of the central zone are atrophic or in part necrotic, or show varying stages of fatty degeneration, cloudy swelling, and necrosis. In some lobules the liver cells of the central zone have completely disappeared. In the majority of lobules there is an endothelial or fibroblastic proliferation in the capillaries of the central zone. This is most marked in those areas showing the most pronounced atrophy or necrosis of the liver cells. The liver rods present a more decided radiating appearance than usual, and in many places appear as if elongated by pressure. The periportal islands of connective tissue are relatively increased in number, the connective tissue in the majority being of a hyaline char-

acter and presenting no infiltration. The fine reticulum of the lobules is everywhere increased, but particularly in the areas of most marked atrophy, where it replaces in part the liver cells. In many areas it is very cellular and presents an endothelial or fibroblastic proliferation. With Van Gieson's stain the greater part of the newly formed reticulum stains a light red. By the same stain heavier lines and strands of connective tissue may be seen in places extending into the lobule. Small focal necroses are found scattered throughout the sections; the necrotic areas showing the same endothelial or fibroblastic hyperplasia as seen in the central part of the lobules. Hypertrophic liver cells are also found throughout the lobules. The central veins and branches of the hepatic veins are greatly dilated and their walls thickened. Giant cells are found in the capillaries; they resemble bone-marrow giant cells and are similar to those found in the lungs and spleen, but are not nearly so numerous as in those organs. In many sections the only changes are those of a chronic passive congestion.

Sections taken from the yellowish, elevated area in the middle of the right lobe show that the liver-lobules are larger and the liver cells are increased in size, the blood capillaries being nearly obliterated. Many of the liver-cell nuclei are very large and stain deeply. In Glisson's capsule there is a marked small-celled infiltration and proliferation of the connective tissue and the smaller bile-ducts. The periportal connective tissue is somewhat increased and the finer reticulum of the lobule likewise shows an increase. The central congestion and atrophy are less marked in this portion.

The liver as a whole presents the picture of a chronic passive congestion and atrophy of the central portion of the lobule, with an increase of the reticulum of the lobules, chiefly central, but also periportal. This increase is in many places due to a fibroblastic proliferation. In some areas these changes are so marked as to warrant their interpretation as a beginning stage of cirrhosis, in part central and in part periportal.

Portal Vein. Sections of the wall show sclerotic changes and calcification, involving chiefly the media. No evidence of recent phlebitis. Branches of portal show similar conditions. Fresh thrombi in all branches.

Stomach and intestines present the picture of a chronic passive congestion with atrophy and chronic catarrhal inflammation.

Pancreas shows decrease in size of lobules and a moderate fatty infiltration. Near the middle of the body of organ a small adenoma the size of a pea was found. This consists of tubules lined with simple columnar cells, the majority showing mucoid degeneration. Pancreatic veins greatly dilated, and walls thickened.

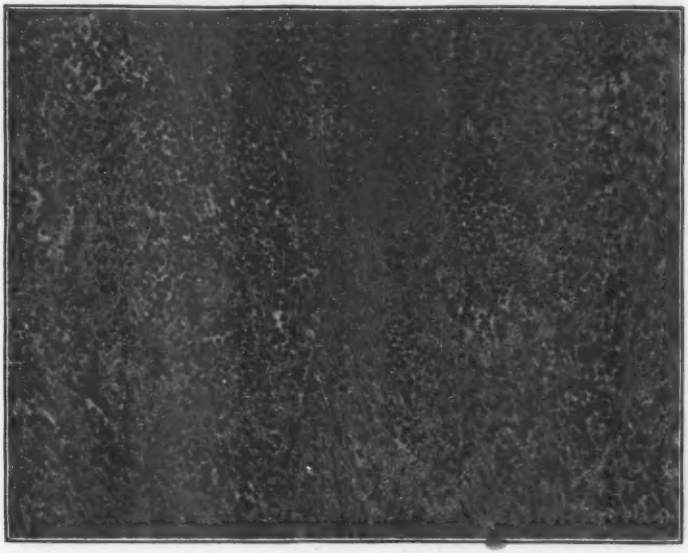
Omentum. Congested and œdematous, and showing a marked small-celled infiltration and fibroblastic proliferation, hemorrhage, etc. Surface covered with a fibrinous exudate.

Mesentery. Peritoneal surface shows patches of fibrin. Very slight small-celled infiltration near surface. The necrosed patches on upper surface correspond to small areas of anæmic necrosis caused by the thrombosis of small mesenteric vessels. Mesenteric veins greatly dilated and sclerotic. Throughout the root of the mesentery are small calcareous nodules surrounded by areas of lymphoid cells. Other collections of lymphoid cells surround small concentric hyaline masses; others surround small arterioles showing hyaline thickening of the

intima, sometimes arranged in concentric laminæ. As all stages of transition can be found between the calcareous and hyaline nodules and the hyaline vessels with proliferating endothelium, it seems probable that the former represent a latter stage of the process. The cells surrounding the masses are of the character of lymphocytes, they lie in a fine reticulum containing small bloodvessels, and some contain typical germ-centres. They are, therefore, regarded as representing newly formed lymphoid tissue. Similar formations are found throughout the retroperitoneal fat.

Mesenteric Glands. Atrophic, and contain hyaline areas, some of which show calcification. Vessels congested. Stroma increased. Few germ-centres seen. Numerous phagocytes in sinuses.

FIG. 4.



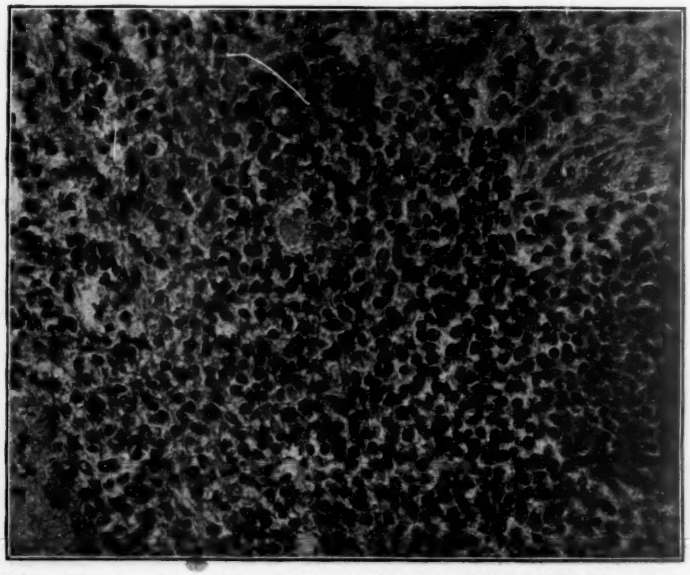
Spleen of Case I. Area showing advanced fibrous hyperplasia of reticulum of pulp. × 175.

Kidneys. Somewhat contracted, the number of glomeruli in a field being greatly increased. Many hyaline and calcified glomeruli are found, as well as numerous cystic ones. Connective-tissue stroma increased in areas. Many of the tubules contain hyaline casts. Bloodvessels congested. No giant cells found in kidney capillaries.

Retroperitoneal Glands. The red spleen-like glands on section are hæmolymph nodes, closely corresponding in structure to that of the normal spleen, and are of the type known as hæmal glands, possessing no lymph vessels or sinuses. They are surrounded by a very thick capsule from which trabeculæ containing unstriped muscle run into the gland. No definite blood sinuses are present; the greater part of the gland structure consists of a pulp-like reticulum and blood spaces lined

by endothelium. Lymphoid cells lie in the meshes of the reticulum. Small scattered collections of lymphoid cells suggest follicles or germ centres, but these lack the true vascular relations of splenic follicles. The blood spaces are filled with blood, and occasionally bone-marrow giant cells are found. An unusual number of pigment-containing phagocytes are present in the larger blood spaces. With Mallory's reticulum stain the reticulum appears as a rather coarse, deeply-blue network enclosing spaces filled with blood. The resemblance to normal spleen is so close that the glands no doubt would be classed as accessory spleens. They resemble very closely the spleen-like hæmolymph glands of the hog. The ordinary lymphatic glands of this region are atrophic,

FIG. 5.



Spleen of Case I. Area showing early stage of endothelial and fibrous hyperplasia. × 175.

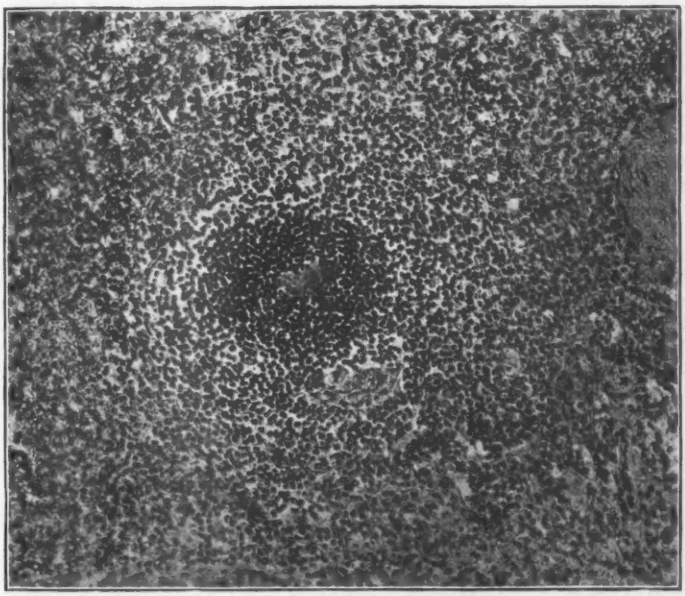
present a great increase of reticulum, with atrophy of lymphoid tissue and almost complete absence of germ-centres. The lymph sinuses are filled with endothelial cells, many of which contain blood pigment. Cords of newly formed lymphoid tissue corresponding to those in the mesentery lie throughout the retroperitoneal fat. The hæmolymph nodes that contain both blood and lymphatic systems do not show the spleen-like hyperplasia seen in the pure hæmal glands.

Mediastinal and Cervical Glands. These are atrophic, congested, and are more or less anthracosed.

Bone-marrow. The red marrow is of a lymphoid type, contains an increased number of normoblasts, and very few giant cells. Numerous phagocytes containing blood pigment are present.

Spleen. (Removed at operation by Dr. Nancrede, November 6, 1899.) Nearly a perfect oval in shape. It measures 23 x 13 x 9 cm., and weighs 1536 grams. Its capsule is greatly thickened and covered with fine stringy adhesions which are more numerous in the region of the hilum. The enlargement of the organ is most marked in the upper two-thirds, the thickness diminishing below. On the anterior margin there are two notches at the lower third, the lower one of these extending almost through the lower part of the organ and forming a shelf-like depression. In the hilum there is a small accessory spleen. On section the spleen bleeds freely. The pulp is a pale red and of firm consistence.

FIG. 6.



Spleen of Case I. Diffuse fibrous and endothelial hyperplasia of pulp. Hyaline mass in follicle. × 175.

The stroma is greatly increased. Splenic follicles cannot be seen. On exposure to the air no Charcot-Leyden crystals were formed on the surface.

The organ was received warm from the surgeon's hands. Portions were fixed at once in Flemming's solution, Müller's, mercuric chloride, alcohol, and formalin.

Microscopic. The capsule is greatly thickened, consisting of a rather dense hyaline connective tissue. It is covered with partially or wholly organized deposits of fibrin. The trabeculæ are thickened to a less extent and are much farther apart than normally. The lymphoid tissue of the pulp is scanty, the pulp being replaced largely by a dense

fibrous network enclosing the enlarged blood spaces. These are lined and in many cases partly filled by large endothelial or reticular cells having one or more lightly staining nuclei, and in the majority of cases nucleoli. The fibrous tissue represents a hyperplasia of the reticulum of the pulp, the endothelial cells lining the blood spaces apparently taking part in the formation of the new fibrous tissue. In many places, particularly beneath the capsule, it is more dense and hyaline, and the blood spaces are smaller. In the denser areas the fibrous tissue stains deep red with Van Gieson's. In such areas the lymphocytes are very scanty and the endothelial cells are not so numerous. In other portions of the pulp the fibrous tissue is less dense, more cellular, stains lighter with Van Gieson's, and presents a more fibroblastic character. In such areas the blood spaces are much enlarged and the number of lymphocytes and endothelial cells much greater. The latter are found not only in the spaces, but also between the fibrillæ of the new connective tissue. Occasional giant cells resembling those of the bone-marrow are found in the blood spaces; they are similar to those found in the lungs and liver, and are probably to be regarded as emboli. There is no evidence of their origin *in situ*. Occasional nucleated red blood cells are found in the blood of the pulp spaces, but neither the ordinary stains nor the triacid stain, etc., show any peculiar cells, myelocytes, or other bone-marrow elements to be present. In the neighborhood of the trabeculæ there are found occasional small collections of densely packed lymphoid cells resembling follicles. A thickened hyaline arteriole may be seen in the central portion of some of these collections of cells; others surround small hyaline masses. The bloodvessels of the organ are very prominent, being greatly dilated and showing marked thickening of the walls. Small deposits of blood pigment (hæmosiderin) are found about some of the trabeculæ, and throughout the pulp spaces occasional endothelial phagocytes containing pigment are seen.

The study of many sections taken from different parts of the spleen leads to the conclusion that the conditions here represent a chronic passive congestion, with great enlargement of the pulp spaces, a coincident or secondary fibrous hyperplasia of the pulp reticulum dependent upon or associated with a hyperplasia of the reticular or endothelial cells lining the spaces. In connection with these changes there is a secondary atrophy of the lymphoid tissue of the pulp and of the follicles. As a result of such atrophy and destruction of the splenic parenchyma the splenic function of hæmolysis is gradually lost. The spleen-like hyperplasia of the hæmal glands, the new formation of lymphoid tissue throughout the root of the mesentery, and the retroperitoneal fat are regarded as compensatory in character.

Summary of Pathology. The most striking feature of the case is the marked fibrous hyperplasia of the spleen. That this cannot be secondary to the liver condition is shown by the marked differences in degree and character of the hepatic and splenic conditions respectively. The splenic condition is, without doubt, much the older condition, as seen by the greater abundance of the fibrous tissue, its greater density and more hyaline character, and its difference in reaction in areas to Van Gieson's stain; moreover, the atrophic condition of the splenic

parenchyma is more marked than that of the liver. The process in the liver is interpreted as a chronic passive congestion and atrophy, followed, secondarily, by an increase of the finer reticulum of the lobules. In several areas, notably in the middle of the right lobe, there is also a beginning periportal increase of connective tissue with hyperplasia of the smaller bile-ducts. This beginning hyperplasia may be regarded as a very early stage or beginning of a cirrhosis, and is evidently of much more recent occurrence than the splenic condition.

In explanation of the congestion and fibrosis of the spleen, the stenosis and calcification of the portal vein may be regarded as an etiological factor. That the changes in the wall of the vein at the junction of the splenic and superior mesenteric veins represent an old and not a recent process is shown by the hyaline thickening of the wall and of the fibrous septa and the plates of calcification. These changes must be taken as evidences of an old thrombosis and pylephlebitis, with subsequent organization, contraction, stenosis, and calcification. It is possible that the stasis in the splenic vein and the damming back of the blood upon the spleen have caused the atrophy and the progressive fibrosis seen in that organ. On the other hand, it is also possible that the splenic condition is primary, and the condition of the portal vein may be purely coincidental or may result from a toxic condition of the blood in the splenic vein dependent upon the disturbed splenic function, or upon a general intoxication, or one affecting the portal area primarily. The changes in the hæmolymph nodes and the new formation of lymphoid tissue are regarded as compensatory for the lost splenic function. It is also probable that a similar compensation is present in the bone-marrow, as shown by the changes described above. The anæmia may be regarded as due either to an excessive destruction of red blood cells by the endothelial phagocytes of the portions of the spleen still exercising the function of hæmolysis, in connection with an excessive activity of endothelial phagocytes in the hæmolymph nodes and bone-marrow, or to a toxic hæmolysis, or to a disturbance of balance between the destruction of hæmoglobin and the elaboration of the products resulting from such destruction. From a careful study of the case we have arrived at the following:

PATHOLOGICAL DIAGNOSIS. Fibrosis of the spleen (*splenomegaly*), with beginning fibrosis of liver (*early stage of Banti's disease*); stenosis and calcification of portal vein, with extreme passive congestion of radicles of portal, particularly of splenic vein; chronic passive congestion of gastrointestinal tract, lungs, etc.; compensatory hyperplasia of hæmolymph nodes and new formation of lymphoid tissue in retroperitoneal fat; increase of lymphoid marrow; acute fibrinous peritonitis following splenectomy; embolism of bone-marrow giant cells; general sclerosis and atrophy; secondary anæmia; cholelithiasis.

CASE II.—Mrs. S., aged fifty years, American, died 12.05 P.M., December 11, 1902. Autopsy by Dr. Warthin at 1.30 P.M., December 11, 1902.

Autopsy Protocol. Cadaver of slender build; no anomalies; no deformities. Right inguinal hernia. Abdomen on level of ribs. In the median line, between the umbilicus and pubes, there is a laparotomy scar 14 cm. long, which is entirely healed with the exception of a sinus about 1 cm. long at about the middle of the scar. Pus exudes from the opening, which communicates by a fistulous tract with an abscess cavity in the abdominal wall.

Skin is pale, hair negative, teeth artificial. Over the back there is pale hypostasis. Transfusion punctures in pectoral regions. The muscles are greatly emaciated; on section they are deep brown and dry. There is no rigor mortis. The panniculus is of a fair amount. Œdema is present over both ankles. Body heat is present.

Brain and spinal cord not examined.

The diaphragm is at the third intercostal space on both sides.

Mammæ atrophic. In the adipose tissue of the right there is a firm swelling about the size of a walnut, consisting of hyperæmic and œdematous fat tissue (transfusion area).

Apex of heart is behind the fourth rib, half-way between the left parasternal and nipple lines. The *mediastinal fat* is abundant and of an orange color; contains no enlarged glands. In the *thymus region* there is an abundance of orange-colored fat. No remains of thymus.

No fluid in *pleural cavities*. The left lung is adherent throughout, the adhesions being very dense over the base, where they have to be cut. Right lung is adherent by easily torn adhesions at apex and over the posterior surface and at the base; the adhesions here not so firm as on left side.

The *pericardial sac* is not tense; contains no fluid. Surfaces of pericardium are moist and shining.

The heart is much smaller than cadaver's right fist; weighs 272 grams, and measures 13 x 9.5 x 5 cm. There is a small, irregular, tendinous spot on the posterior wall of left ventricle; another near the apex, and one on the anterior wall of right ventricle. The subepicardial fat is increased. Heart muscle is pale brown, fairly firm. The right auricle contains currant-jelly and mixed clots and plum-colored fluid blood. The wall of the right ventricle measures 5 mm., 3 mm. of which are of fat. Right ventricle is empty. The left ventricle is empty, wall contracted; measures 20 mm. The left auricle contains plum-colored fluid blood and small red jelly clots.

The *mitral valve* barely admits two fingers. Circumference of orifice, 8 cm. Flaps are thickened along proximal edges. Tricuspid barely admits three fingers; orifice measures 11 cm.; flaps negative. Pulmonary barely admits thumb; orifice measures 6.5 cm.; flaps negative; aortic admits thumb easily; orifice 7 cm. in circumference; slight thickening of central segment.

The *aorta* shows beginning sclerosis, most marked about the coronary orifices. The descending branch of the left coronary artery is moderately sclerosed.

The *left lung* weighs 216 grams, and measures 17 x 13 x 3 cm. It is collapsed, pale, moderately anthracosed, and the pleura is covered with the remains of adhesions. On section the lung is pale, dry, almost

bloodless, and yields almost no fluid on pressure. Beneath the pleura, particularly over the lower lobe, there are scattered a few dark areas of small size, which are firm on pressure.

The right lung is more voluminous and contains more air than the left. It weighs 256 grams, and measures 19 x 12 x 4 cm. On section it is more pink and moist over the middle and lower lobes; otherwise similar.

Bronchi negative. Bronchial glands moderately anthracosed. Pulmonary vessels contain large ante-mortem mixed clots and post-mortem jelly clots.

Thoracic aorta measures 5 cm. in circumference; contains small jelly and mixed clots. The azygos veins are greatly dilated.

FIG. 7.

Low-power view of liver of Case II., showing picture of advanced cirrhosis. × 45.

Thoracic duct is distended with clear fluid.

Thoracic portion of œsophagus contains a brownish fluid. The lower third shows marked varices; the œsophageal veins and their anastomoses with the coronary veins of the stomach are markedly dilated. There are no evidences of hemorrhage from the œsophageal mucosa. No erosions of the varices.

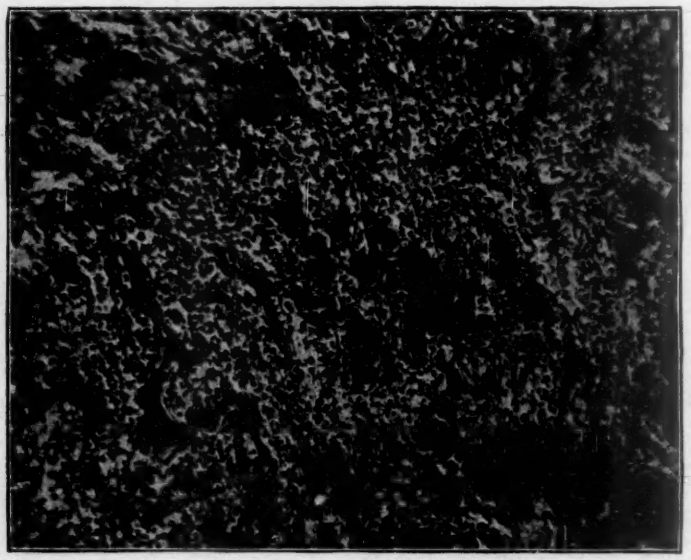
Mouth, pharynx, and *larynx* not examined. The cervical portion of the œsophagus contains a brownish fluid. The thyroid is small and of firm consistence. On section it is pale and more brown than normal.

Cervical Hæmolymph Nodes. In the deep cervical tissues numerous reddish, spleen-like glands resembling hæmolymph glands are found. They are larger and appear much more numerous than normally.

Cervical vessels and nerves negative.

Abdomen. The *omentum* is adherent to the abdominal wall in the neighborhood of the umbilicus by easily torn adhesions, between which there is a fibrinopurulent exudate. The lower border is adherent to the anterior abdominal wall just above the pubes. The intestinal coils present scattered fresh fibrinous adhesions, particularly on the left side. From between the adhesions one and a half litres of turbid fluid containing many fibrin flakes are obtained, the greater part being taken from the left side below the spleen. The adhesions are strongest in the neighborhood of the incision. In the abdominal wall at this point there is a fistulous tract extending through the rectus muscle as far as the

FIG. 8.



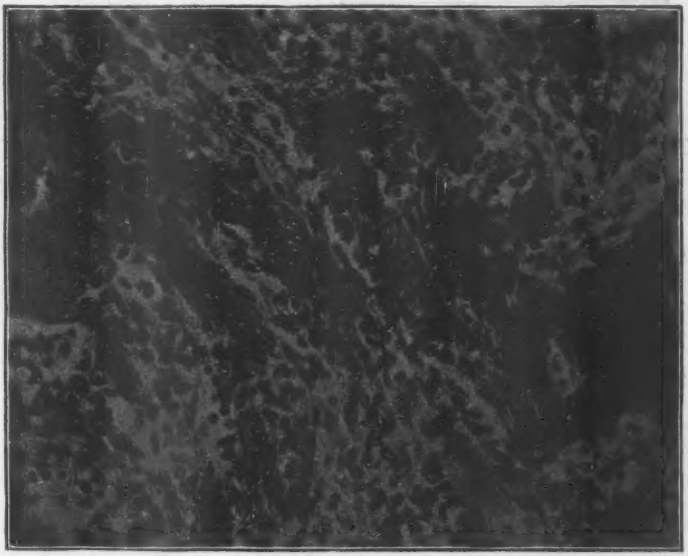
Spleen of Case II. Fibrous hyperplasia of reticulum, with hyaline change. Dark areas = hyaline area stained deep red with Van Gieson's. No follicles. × 175.

pubes, and communicating with the external opening in the laparotomy wound. Between the adhesions there is a collection of pus. The intestines in this region are firmly adherent to each other and to the abdominal wall. In one of the loops there is a darkened, thin, and softened area, but no perforation. The pelvis is filled with coils of small intestine which are adherent to the sigmoid and rectum. Between these adhesions there are about 50 c.c. of fluid similar to that in the abdominal cavity, but more turbid, and containing more fibrin. The transverse colon and cæcum are moderately distended; the former is S-shaped, the lowest part two fingers' breadth above the umbilicus. The *liver* is high and very small, the lower edge in the right parasternal line being three finger breadths above the edge of ribs and just at the tip

of the ensiform in the median line. The left border is two fingers' breadth to the left of the left sternal line. The *spleen* is greatly enlarged, filling in the entire left hypochondrium and projecting a hand's breadth below the edge of the ribs. It is adherent to the diaphragm by several easily torn adhesions. The lower border of the stomach is three fingers' breadth below the ensiform. There are easily torn adhesions between the stomach, omentum, and hilum of spleen. No accessory spleen found in gastrosplenic omentum.

Spleen weighs 944 grams, and measures 19 x 14 x 7 cm. It is triangular in shape, broader at upper pole, the enlargement involving chiefly the upper third. The upper pole is slightly recurved. There is one deep

FIG. 9.



Spleen of Case II. Higher power view of Fig. 8. Fibrous hyperplasia of splenic reticulum, with hyaline change. Dark areas = hyaline areas staining deep red with Van Gieson's. × 350.

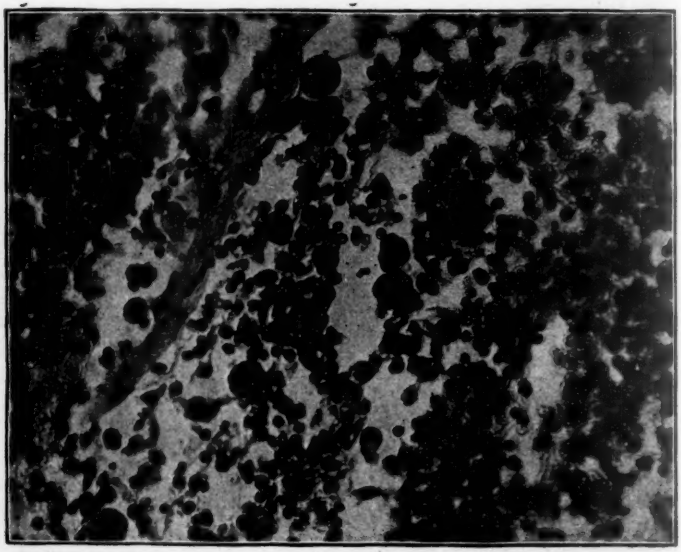
notch at about the middle of the anterior border, extending nearly halfway across the organ. The capsule is thickened, particularly along the anterior margin, where there are flattened hyaline thickenings and the remains of stringy adhesions. The organ flattens somewhat, but is of increased consistence. On section it is firm and tough, creaks to the knife, pale, grayish-blue red, almost homogeneous, and shining, semitranslucent, but slightly granular in areas. Contains but little blood. The follicles are greatly diminished in number and in size—only a few can be seen. There is a diffuse fibroid hyperplasia of the reticulum of the pulp. The vessels at the hilum appear normal; veins collapsed.

Adrenals well preserved; no pathological changes.

Left Kidney. Fatty capsule very rich in fat. Fibrous capsule strips easily; shape about normal; less plump than the right one; weighs 120 grams; measures 10 x 6 x 3 cm. Small, yellowish areas on the posterior surface. These are neither elevated nor depressed. On section they are found to extend through the cortex and medullary pyramids. In other respects resembles right kidney.

Right Kidney. Fatty capsule very rich in fat. Fibrous capsule strips easily. Surface of kidney smooth, mottled brownish-red and yellow; weighs 104 grams; measures 8½ x 5½ x 3 cm. Several yellowish spots, size of a pea, seen over the surface. On section cut surface is pale brownish-red, containing small yellowish areas size of a pepper-corn,

FIG. 10.



Portion of a sinus of retroperitoneal hæmolymph node from Case II. Large phagocytes filled with red blood cells, the outlines of the latter being just visible. Free blood cells in meshes of blood sinus. × 175.

and is more translucent than normal. Cortex measures 2 to 3 mm. Outlines of labyrinths and medullary rays well preserved. Vessels anæmic; consistence firm; subpelvic fat increased; pelvis negative.

Ureters negative.

Bladder distended with cloudy urine. Mucosa negative.

Duodenum contains plum-colored, bloody fluid, with strong odor of H_2S. In this fluid are four large currant-jelly clots, weighing 12.2, 10.8, 7.3, and 6.1 grams respectively. Mucosa anæmic, and presents no erosions or evidences of hemorrhage.

Bile Passages. Patent.

Small Intestine. The jejunum and ileum contain fluid similar to that found in duodenum, with yellowish clots. Toward the lower end of the

ileum the fluid becomes thicker and darker in color. The mucosa is very anæmic. No evidences of hemorrhage.

Appendix. Firm adhesions containing pus about the appendix. Lumen negative.

Large Intestine. Entirely filled with tar-like material. About the middle of transverse colon there is a small polyp. Mucosa is very anæmic. No evidences of hemorrhage. The *rectum* is filled with a black, tarry, tenacious material adhering to the mucosa. In the latter are the scars of healed ulcers. No evidences of hemorrhage. Hemorrhoidal veins greatly enlarged, but collapsed. No evidences of hemorrhage from these.

Stomach. Contains 750 c.c. of a thick, black fluid, composed chiefly of red blood cells and a few small red clots. The mucosa is thickened and covered with a thick, brownish mucus. About 14 cm. below the cardiac orifice there is in the posterior wall an erosion in the mucosa, having undermined edges, and about 3 mm. in diameter. Beneath this and under the neighboring mucosa there is a space about 2 cm. in diameter, filled with a fresh blood clot, and communicating with a ruptured vein in the posterior wall, a greatly dilated branch of the coronary veins. This is undoubtedly the source of the hemorrhage. All of the veins of the stomach wall are greatly dilated and tortuous, but are for the chief part collapsed and empty. The coronary veins in particular show a marked dilatation.

Liver. Small, about size of a child's head, nearly round, but irregular. Weighs 1856 grams; its greatest dimensions are 23 x 17 x 11 cm. It is adherent to the diaphragm by very firm and tough adhesions. The surface is very irregular and nodular, particularly along the lower edge and near the ligaments. It contains numerous small hyaline areas and small, translucent, glistening, brownish points, varying in size from that of a pinpoint to that of a pinhead. These are most numerous along the lower edge. Over the upper surface of the organ the capsule is 0.5 cm. thick, and firmly adherent to the diaphragm. The consistence is very firm; creaks on cutting. On section Glisson's capsule is greatly thickened in areas, the cut surface being mottled whitish and brown, the white areas corresponding to the dense hyaline connective tissue of the thickened Glisson's capsule. The increase of Glisson's capsule is, however, very irregular, being most marked in the central portion of the right lobe and beneath the external capsule. There appears to be a diffuse increase of connective tissue throughout the organ. The cut surface is slightly granular, the liver tissue showing as irregular islands surrounded by connective tissue. Surface is more translucent than normal. The outlines of the lobules cannot be made out. The branches of the hepatic vein are dilated, those of the portal nearly obliterated by the periportal increase of connective tissue. No icterus.

Gall-bladder is very small; size of a pigeon's egg. It is retracted above the edge of the liver. Tissues of the wall are thickened. It contains a very thick, glairy, brownish-like bile; no stones.

Portal Vein. Just above the junction of the splenic and superior mesenteric veins the wall of the portal vein is thickened and contains calcareous plates. The lumen is stenosed, barely admitting tip of lead-pencil. The wall is irregularly thickened and shows lines of contraction radiating from the areas of calcification. The lumen is filled with a

recent mixed thrombus, chiefly white, firm in consistency, and extending to the portal of the liver and into the smaller portal branches within the liver. No evidences of organization of the thrombus. All the radicles of the portal are enlarged and show thickenend walls, the splenic vein in particular.

Pancreas. Smaller than normal. Lobules atrophic. Fatty infiltration between the lobules. Throughout the organ there are numerous whitish areas of fat-necrosis in the interlobular fat. These are most marked along the upper border. In the great omentum about a hand's breadth below the greater curvature there is a red body the size of a cherry, embedded in the omental fat. It is rather soft and fluctuating, resembling a congested hæmolymph node. On section it shows a small, firm, whitish nodule resembling epithelial tissue and suggesting pancreatic tissue, the remaining portion being made up of reddish, lymphoid tissue and a small abscess cavity containing a brownish, pus-like material. Small areas of fat-necrosis are found in the fat-tissue near by.

Mesentery. Mesenteric glands not enlarged. No hæmolymph nodes found except in root, where there is a great number of reddish and bluish glands embedded in the mesenteric fat, probably hæmolymph nodes.

Retroperitoneal Glands. Over 250 red, spleen-like glands found in the root of mesentery, retroperitoneal fat, about the renal vessels, behind and along the great abdominal vessels, and particularly along the brim of the pelvis. Along the left common iliac there is one 4 cm. long and as thick as a lead-pencil. A similar cord lies along the vena cava. The majority of the red glands are the size of a bean; others vary in size from that of a pinhead to that of a bean. On section they are firm, bluish-red, and spleen-like; only a few show dilated sinuses and a softer consistence.

External Genitals. Negative.

Uterus and Appendages. Absent. (Removed at operation.)

Site of operation covered with fibrinopurulent exudate. About 50 c.c. of turbid fluid in pelvis.

Bone-marrow. Tibias, opened longitudinally, show no lymphoid changes. Red marrow in sternum and ribs hyperplastic.

Peripheral Lymph Glands. Prominent, but not enlarged.

Peripheral Bloodvessels. Femoral veins filled with recent thrombi; the left completely occluded by a clot of firmer consistency than that found in the right femoral. Lumbar and azygos veins greatly enlarged. (Material taken from all organs and tissues fixed in Flemming's, Müller's, Zenker's, mercuric chloride, alcohol, and formalin. Embedded in paraffin; stained in ordinary and specific stains.)

Microscopic Examination. Heart. Sections of heart muscle show brown atrophy. Fatty infiltration of right ventricle.

Lungs. Sections show atrophy and sclerosis of bloodvessels. Great numbers of bone-marrow giant cells in capillaries.

Spleen. Marked fibrosis of the reticulum of the pulp, with hyaline change of the newly formed connective tissue, the blood spaces being outlined by hyaline cords of varying thickness. The process is fairly uniform throughout the entire organ, although denser and more hyaline areas, with wholly or partially obliterated blood spaces, are seen in portions, particularly near the external capsule. The blood spaces are very prominent, and many greatly enlarged, some areas being cavernous.

The lymphoid tissue of the pulp is very atrophic, and only small, scattered collections of cells resembling follicles are seen. The majority of these contain hyaline masses. The blood spaces are lined by large, hypertrophic, endothelial cells, but these do not show any evidences of hyperplasia into the spaces. A few giant cells resembling those of the bone-marrow seen in the blood spaces (embolism?); a few normoblasts are also found. No unusual forms of cells observed in sections stained by special methods (triacid, polychrome, methylene blue, etc.). All the bloodvessels of the spleen are enlarged and show marked sclerotic changes and numerous nodules of calcification. A deposit of lime-salts is also found in some of the hyaline masses of the follicles. Testing of many sections for iron with potassium ferrocyanide and hydrochloric acid entirely negative. No phagocytes containing blood pigment seen, and no free blood pigment.

Liver. Sections taken from different parts of the liver show that the greater part of the organ presents the picture of an atrophic cirrhosis. Glisson's capsule is greatly increased, and there is also a growth of connective tissue into the lobule. The periportal increase of connective tissue is, however, very irregular; in some areas it is very marked, in others it is but slight, small areas showing no cirrhotic changes. In other areas there is a central increase of connective tissue. The greater part of the lobules show central dilatation of the capillaries and atrophy of liver cells. Much of the connective tissue, particularly toward the surface of the organ, is hyaline and scar-like; in those areas showing less change the new tissue has more of a fibroblastic character and contains more small cells. There is a slight increase of the small bile-ducts. Liver cells are atrophic; those of the central portion of the lobule contain a very small amount of hæmatoidin. Iron test is entirely negative.

Pancreas. Sections show a fatty atrophy. Vessels are sclerosed. There is a small-celled infiltration of the interlobular fat, and localized areas of fat necrosis with small hemorrhages are present.

Body from Omentum. Sections show the red body found in the omentum to consist of three separate structures: A hæmolymph node showing great numbers of phagocytes containing red cells in its blood sinuses; an accessory pancreas containing all the characteristics of pancreatic tissue, acini, and areas of Langerhans. There is an increase of the stroma of the parenchymatous portion as well as of the islands, and an infiltration of fat-cells. No large duct leading from this accessory pancreas can be found, but a small duct can be traced leading into the third structure of the mass—an abscess cavity filled with a pus-like material, and having a wall of granulation tissue in which there are many large, thin-walled vessels. Small groups of necrosed fat-cells are found in the neighboring tissue.

Stomach and Intestine. Sections show a chronic passive congestion with catarrhal inflammation.

Kidneys. Sections show atrophy, sclerosis of vessels, and localized areas of interstitial inflammation. Convoluted tubules show moderate cloudy swelling. Iron test wholly negative.

Hæmolymph Nodes. These constitute one of the most interesting features of the case, judged from the pathological standpoint. The changes may be summed up as follows: All the red glands examined show the structure of hæmolymph nodes in varying stages of hyperplasia. They contain but few germ-centres. The blood sinuses are all

very prominent and large, but the majority are only partly filled with blood. In some glands the sinuses are distended. They contain also great numbers of large hyaline cells having a yellowish or brownish color, and staining deep red with eosin. Some of the sinuses are completely filled with these cells. With the higher powers these cells are found to be phagocytes completely filled with red cells, or with the products of the disintegration of red cells. The nucleus of the phagocytic cell is usually pushed to one side, and shows more or less karyorrhexis. The outlines of the red cells can be distinctly seen in the phagocytes, and in many cases appear unchanged. In other phagocytes only hyaline droplets staining deeply with eosin or a brownish pigment are seen. The majority of the cells give a strong iron reaction with ferrocyanide and hydrochloric acid. In some there is a yellowish pigment which does not respond to the iron test (hæmatoidin?). Great numbers of mononuclear eosinophiles are found in some of the glands. Normoblasts and giant cells resembling those of the bone-marrow are found in large numbers in the blood sinuses. With the triacid stain numerous cells resembling myelocytes (neutrophile granulation and large excentric nuclei) are also found in the blood sinuses. In many of the retroperitoneal hæmolymph nodes there are also large masses of hyalin which occupy the lymphoid areas between the blood sinuses. Many of these show calcification. Encapsulated caseous tubercle found in one gland. In the sections treated with ferrocyanide and hydrochloric acid the areas of calcification give a marked iron reaction, which is diffuse or very finely granular. A similar reaction is observed in many of the hyaline areas in which calcification has not taken place. The appearances suggest some connection between the hyaline masses and the blood destruction.

Lymphatic Glands. Atrophic; show marked hyaline change.

Bone-marrow. The normoblasts are increased, and the giant cells greatly diminished in number. The reticulum of the red marrow appears hyperplastic and the blood spaces smaller. In the blood spaces and in the reticulum are great numbers of pigmented phagocytes, as in the hæmolymph nodes. Free pigment is also present. The iron test shows the pigment to be hæmosiderin.

Tumor of Uterus (removed at operation). The tumor was found to be nearly completely calcified. After decalcification of portions sections were obtained showing it to be a myofibroma, with increase of the connective tissue, atrophy of the muscle, hyaline change, and calcification. Traces of atrophic muscle found throughout the dense hyaline tissue between the masses of calcification. Very few nuclei found in the hyaline connective tissue.

Summary of Pathology. The immediate cause of death is the anæmia resulting from the hemorrhage from the eroded varix in the stomach wall. The dilatation of the stomach and splenic veins is the result of the portal obstruction. For the latter two etiological factors are present, the stenosis and calcification of the portal vein and the periportal cirrhosis. It is impossible to decide the relative part played by these factors. The most important question is the relation of the splenic condition to that of the liver. It is, however, impossible to decide positively which is the primary condition, the splenic fibrosis or the cirrhosis of the liver. Because of the more advanced stage of the

splenic fibrosis, the dense hyaline character of the new tissue formed in the walls of the pulp spaces, the areas of hyaline and calcification, the greater atrophy of splenic parenchyma, etc., it seems very probable that the splenic process is older than that of the liver. If this is the case the question also arises here as to the relationship between the changes in the spleen and those in the portal vein. Is the splenic fibrosis primary, or is it secondary to the disturbance of the splenic circulation following thrombosis and the resulting stenosis of the portal? This question, unfortunately, cannot be positively answered. The portal condition is, undoubtedly, the result of an old pylephlebitis and thrombosis, but it is impossible to determine if it preceded the splenic condition or is secondary to some toxic condition of the portal blood, dependent, possibly, upon a primary change in the spleen or upon a general toxæmia.

The changes in the hæmolymph nodes are to be interpreted as compensatory for the lost splenic function. The complete absence of blood pigment or of phagocytes in the spleen may explain the great excess of hæmolysis occurring in the hæmolymph nodes and bone-marrow. The evidences of phagocytic hæmolysis in the hæmolymph nodes presented by this case show a much greater blood destruction than that occurring in these glands in any of the cases of pernicious anæmia observed by the writer. In spite of so great a hæmolysis no hæmosiderin is found outside of these glands and the bone-marrow. Iron tests of liver, spleen, and kidneys do not show a trace of iron. The hæmolysis is therefore different from that occurring in pernicious anæmia, and is most probably not a *toxic hæmolysis*, but is to be interpreted as a *compensatory* one. The anæmia may then be explained as due to an excess of hæmolymphatic hæmolysis over that of the normal splenic hæmolysis, or to a disturbance in the elaboration of the products of blood destruction.

There is no histological evidence of a destruction of red cells in the general circulation, as is the case in pernicious anæmia.

From the study of the case we have arrived at the following:

Pathological Diagnosis. Splenic fibrosis with cirrhosis of the liver (late stage of Banti's disease), gastric hemorrhage, anæmia, stenosis, and calcification of portal vein, chronic passive congestion of portal system, ascites, peritonitis, hyperplasia of hæmolymph nodes with excessive hæmolysis, hyperplasia of red marrow, giant-cell embolism, general atrophy and sclerosis, tuberculosis of retroperitoneal gland, accessory pancreas, fat-necrosis.

Comparison of Cases. Both cases present the following common features:

1. Splenic fibrosis.
2. Hepatic fibrosis.
3. Stenosis and calcification of portal vein.
4. Chronic passive congestion of portal system.

5. Sclerosis of vessels, particularly of portal area.

6. A marked tendency to hyaline change and calcification throughout portal area.

7. Hyperplasia of hæmolymph nodes and bone-marrow and new formation of lymphoid tissue in retroperitoneal fat.

8. Secondary anæmia.

Comparing the two cases the splenic fibrosis in Case I. represents an earlier stage; that in Case II. a much more advanced stage. In Case I. the liver changes are relatively slight, a beginning fibrosis. In Case II. they are marked—an advanced cirrhosis, a cirrhosis, however, which does not present all the features of an ordinary atrophic cirrhosis. The changes in the portal vein in both cases were at the same site and of about the same degree. In Case I. the more irregular character of the vessel wall, the bridging strands of tissue across the lumen of the vein, might indicate a more recent process. The chronic passive congestion of the portal is more marked in Case II. than in Case I., as shown by the greater development of anastomoses and the pronounced varices in œsophagus and stomach leading to the fatal hemorrhage in the former case.

In Case I. the hyperplasia of the hæmolymph nodes is not nearly so marked, and in these glands there are not the evidences of the pronounced hæmolysis seen in Case II. In Case I. the spleen contains some phagocytes and blood pigment. In Case II. none of these were found in the spleen. These differences may also be interpreted as indicating an early stage of the process in Case I. and a later stage in Case II. The pathological findings, therefore, would appear to warrant the diagnosis of early and late stages of Banti's disease.

In both of these cases (unlike some others described under the head of splenic anæmia or Banti's disease) there is no clinical evidence and no positive anatomical proof of the existence of syphilis. On the contrary, the clinical history gave strong arguments against the possibility of this disease.

The important question as to the relationship between the splenic condition and the stenosis of the portal remains to be settled. Is the splenic fibrosis a distinct pathological entity or is it a secondary process? The changes in the spleens of the two cases do not represent any features that could not be explained by a chronic passive congestion of marked degree; that is, it is possible for such a hyperplasia of connective tissue to follow a severe and long-continued passive congestion, as is sometimes the case in the lung, liver, etc. This question, however, cannot be answered by these two cases. It can only be said that there is a possibility that the clinical picture of splenic anæmia or Banti's disease may be caused by a stenosis of the portal vein due to old phlebitis and thrombosis and the resulting stenosis.

On turning to the cases of splenic anæmia reported in the literature under such heading, we find that the condition of the portal vein is not mentioned at all, or so briefly as to convey the impression that the vessel had not been carefully examined. In the older literature there is, however, a number of very suggestive cases similar to the two described above, and reported under the head of "Thrombosis," or "Stenosis of Portal Vein, with Hypertrophy of the Spleen." The descriptions given of these cases, though incomplete and meagre, when judged from modern standpoints of diagnosis, suggest very closely the cases now described as splenic anæmia. Among these is one reported by Sir Andrew Clark (*Trans. London Path. Soc.*, 1867). The patient had suffered previously from "biliousness" and intestinal hemorrhages; died of gastric hemorrhage. Skin sallow, body very fat. Autopsy showed spleen to be three times normal size; liver "thinner" than normal; wall of portal vein contained calcareous plates; in the lumen there was a fresh thrombus.

It is possible that this and some of the similar cases reported in the literature, as well as some of the cases of cirrhosis with changes in the portal vein, may represent the same morbid processes as do our two cases and that they should be placed in the category of splenic anæmia.

Conclusion. The two cases reported above present, clinically and anatomically, the pictures of an early and a late stage of Banti's disease —an older fibrosis of the spleen with a more recent fibrosis of the liver. Both cases present also the condition of stenosis and calcification of the portal vein. Is the latter the primary condition, to which the splenic fibrosis follows secondarily, or is it the result of a portal or general intoxication, to which the splenic and hepatic fibrosis are also due? The solution of this problem must wait upon the observation and investigation of other similar cases. The two reported above, however, may be taken as further evidence in favor of the view that the symptom-complex of splenic anæmia represents a group of varying pathological conditions, the splenic condition being secondary.

DESQUAMATION OF THE SKIN IN TYPHOID FEVER.*

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Desquamation of the skin of a pronounced character is rare in typhoid fever, and is mentioned by but few of the systematic writers

* Read before the Section in Medicine of the College of Physicians of Philadelphia, February 9, 1903.

upon the disease. The great Louis,[1] whom almost nothing escaped, refers to it in the following words: "Desquamation of the epidermis occurred to a greater or less extent in some of the grave cases of the typhoid affection that terminated favorably, even in persons that had no sudamina. Like the sudamina, this desquamation indicated a previous disease of the skin, and I regret very much that I did not look for it oftener." Elisha Bartlett,[2] the first American writer to recognize, in a systematic work, the difference between typhoid and typhus fevers, makes no mention of desquamation. Nothing is said about it in the majority of the French theses upon typhoid fever—and the number of these following Louis' publications is truly astounding. I find a brief reference in one of them, that of Macario,[3] who speaks of desquamation as following sudamina. Harley,[4] in Reynolds' *System*, says that roughness and desquamation of the cuticle, especially of that covering the abdomen, are observed after the cessation of febrile symptoms in severe cases, the desquamation occurring independently of the existence of sudamina, which alone is sufficient to produce it. This is practically a quotation from Murchison,[5] who notes desquamation, in the form of minute, branny scales, as either following sudamina or occurring independently during convalescence. Liebermeister[6] does not refer to desquamation; Dreschfeld,[7] in Allbutt's *System*, says that a desquamation of fine, branny scales is often observed toward the end of the fever or during convalescence. Hutchinson,[8] in Pepper's *System*, alludes to desquamation following sudamina, but makes no mention of its occurring in other circumstances. Strümpell[9] speaks of desquamation; and Osler,[10] in the last edition of his book, states that a branny desquamation is not rare in children, and that occasionally the skin peels off in large flakes. In his extensive observations on typhoid fever in the Johns Hopkins Hospital, Osler[11] noticed a distinct desquamation only four times. Wilson[12] states that slight desquamation is occasionally observed during convalescence; and Chantemesse[13] speaks of furfuraceous desquamation on the lateral aspects of the body, especially in children. In his elaborate monograph on the *Medical Complications of Typhoid Fever*, Hare[14] alludes to desquamation, and states that it is common and that he has frequently seen it. Brannan,[15] in *Twentieth Century Practice*, says that a desquamation of fine, branny scales may occur in typhoid fever independently of sudamina. Both he and Hare assert that the desquamation occasionally follows a scarlet rash or erythema that at times occurs in typhoid fever without any obvious cause.

One of the first to study the subject of desquamation specifically was Weill,[16] of Lyons, who, like myself, was struck with the paucity of references to it in literature, he having succeeded in finding but one, viz., in the thesis of Hutinel. Weill's studies were made principally

upon children, and in 37 cases he found desquamation thirty-three times. He also observed it in adults, but not so frequently. In 1895 Coulon[17] reported a case of scarlatinous desquamation following typhoid fever in a girl aged eleven and a half years.

Stimulated by the observations of Weill, Comby,[18] the well-known French pediatrist, gave attention to the subject of desquamation, and found it in all of 18 cases of typhoid fever in children examined for it. He attributes it to antecedent sudamina. Weill, on the other hand, does not think that it has anything to do with sudamina, but looks upon it as the analogue of the alopecia and the ungual changes that are so common in typhoid fever.

The desquamation occurs on the trunk, the shoulders, and the hips. According to Weill and Comby, it is never observed on the face, the palms, or the soles. This, however, is not strictly correct, as will be seen in the history of my second case. The desquamation begins toward the end of the disease—as a rule, at the commencement of convalescence or a little later—and lasts from several days to several weeks. Weill believes that there is a relation between the form, intensity, and duration of the desquamation and the character of the fever, grave cases being usually accompanied by early, extensive, lamellar, and prolonged desquamation; while in mild types the desquamation occurs later, and is discrete, furfuraceous, and transient. As stated above, Comby considers the desquamation to be a sequel of the sudaminal eruption; he looks upon the latter—erroneously it seems to me—as a critical discharge in the typhoid fever of children, even going so far as to say that it is absent in patients that are going to die.

As in one of Osler's cases, the desquamation may at times be preceded by a diffuse, erythematous blush, the true nature of which is problematical. Very probably it is of septic origin. In the second of my patients, who was under continuous observation, such a rash was not noticed. The first patient was not seen until the stage of desquamation was well advanced.

The question of the infectivity of the scales might be raised; it is hardly likely, however, that they play any part in the epidemiology of the disease.

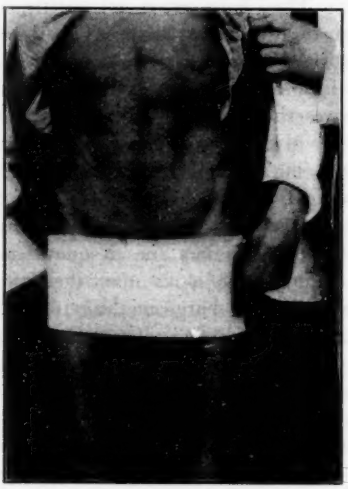
The possibility that vigorous sponging and the bath treatment may be provocative of desquamation suggests itself. I do not think, however, that the rare form presented by my two cases, in which desquamation occurred in large scales, is produced by this agency. In Case II. the desquamation was more marked on the limbs and the abdomen than on the chest, and was quite pronounced on the face—a part not subjected to special hydrotherapeutic measures. A limited furfuraceous scaling might follow in the wake of the bath-treatment, but Comby

emphatically states that the desquamation observed by him bore no relation to bathing.

My own cases are two in number:

The first patient, J. S., aged seventeen years, had a typical attack of typhoid fever. He had been admitted into the inebriate ward of the Philadelphia Hospital and afterward transferred to the ward of Dr. Henry, where I happened to see him. My thanks are due to Dr. Henry for this privilege and for permission to make use of the case.

The patient was admitted on February 11, 1902, the abdomen showing at that time many rose-colored spots. The temperature reached normal on the 28th of the same month. During the height of the fever the patient received two tub-baths and nineteen antipyretic sponges. There are, unfortunately, no notes concerning the desquama-



tion; but the photograph, here reproduced, which was taken during convalescence, shows clearly an extensive formation of fine scales over the abdomen, the thighs, and the upper parts of the legs.

The second patient, S. J. F., a native of Sweden, was admitted to the Philadelphia Hospital on January 23, 1902, in the service of Dr. Tyson, to whom I wish to express my thanks for the opportunity of studying the case. The symptoms were those of a severe type of typhoid fever. The characteristic eruption was present on admission, but was not unusually profuse. There was a marked bronchitis and a high degree of adynamia. Within a few days after admission the rash became more widespread, spots appearing on the arms, the face, and the thighs, and a few being seen also below the knees. The temperature, which had not been high at any time, reached normal on February 12th, about three weeks after the patient's entrance into the hospital, and

probably four weeks after the beginning of the fever. Tub-baths were at no time required, and the patient was sponged for fever but once. The temperature remained normal until March 3d, when, after a severe chill, it rose to 106° F.; there being, at the same time, acute pain in the right side. The signs of pleurisy promptly appeared, and an effusion necessitating aspiration rapidly formed. The patient left the hospital in a fair state of health early in April.

The desquamation of the skin began during convalescence from the typhoid fever; but there is no record of the exact date. It was widespread and in the form of scales, some of which were fully a quarter of an inch long. On the chest and the abdomen the desquamation was most noticeable, but it was scarcely less pronounced on the extremities. It could also be seen distinctly on the forehead, the ears, the nose, the cheeks (especially the malar prominences), and the neck. There was no desquamation in the region of the hairy scalp; nor on the shoulders, the palms, or the soles. On the back it was slight. On the extremities the distribution of the scales was somewhat peculiar. Thus, on the arms it was disposed in the form of narrow, circular bands, from one-eighth to one-quarter of an inch in width. The desquamating circles were almost parallel, with zigzag borders, but in a few places the bands ran into one another. In the interspaces, which were about as wide as the bands themselves, the skin appeared to be normal. The desquamating areas were dead-white in color and somewhat crinkled. On the legs the desquamation was arranged in similar circular stripes, the scaling being just as pronounced there as on the arms. A photograph of this case was taken, but, unfortunately, it does not do justice to the condition.

The patient was exhibited to several sections of students, and all that saw him were struck with the appearance of the skin. I have here a few of the scales, taken at random. They will give an idea of the size. I firmly believe that anyone seeing the patient without knowing anything of his history would have concluded that he had passed through an attack of scarlet fever or of some other exanthem attended with pronounced scaling.

Conclusions. We may distinguish the following varieties of desquamation in typhoid fever:

1. That confined to the roseolar spots. In some instances each spot has a tiny vesicle upon its summit, which quickly passes into a thin, scale-like crust. I myself have never seen such vesicles, but they are mentioned by several good authorities. I have, however, noticed a tiny scale in the areas of the spots.*

2. That appearing as a sequel of sudamina. This is confined to the

* Since writing this I have had a patient at the Polyclinic Hospital in whom a few, but not all, of the rose-colored spots were each capped with a minute vesicle.

areas that have been the seat of the sudaminal eruption, although Louis states that the intervening skin may readily be peeled off. The desquamation is usually furfuraceous, but is sometimes scaly. It occurs upon the trunk and the proximal parts of the limbs, and is never seen upon the distal parts of the extremities or upon the face. It appears in the bathed and in the unbathed.

3. In some instances, as illustrated by the second case here reported, there is, in typhoid fever, an extensive, almost universal desquamation, either furfuraceous or lamellar, which seems to be independent of sudamina, and in all probability is a trophic change analogous to the shedding of the hair. It affects the trunk and the roots of the limbs, and, in rare instances, also the face and the distal parts of the extremities. Usually, but not always, the extent and the intensity of the desquamation bears a relation to the severity of the fever.

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THE DIFFERENTIAL DIAGNOSIS OF TYPHOID FEVER IN ITS EARLIEST STAGES.[1]

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Note.—In the following article the author has drawn extensively on many standard works and also upon the medical journals, both foreign and domestic. He is aware that too great an amount of medical

[1] Read before the Association of Military Surgeons of the United States, at the annual meeting, Boston, Mass., May 19, 1903, and awarded second prize by the Enno Sander Prize Medal Board of Award.

compilation and redundancy is being imposed daily upon the medical public as original work, and has endeavored as far as possible to assemble only those facts which bear directly upon the subject in hand. He has fused his own experiences with those of many other observers and tried to give an impersonal résumé of the whole, endeavoring to treat the subject in its entirety, rather than make the paper an excuse for a dissertation on one particular point.

In the list of references an attempt has been made to mention the name of every author whose work has been quoted, or from whom ideas have been borrowed for this essay. Experiments have not been quoted in detail, as they are at best uninteresting reading, and only the results of such work have been here recorded.

The term "earliest stages," while it limits in a measure the ground to be covered by this essay, is vague and indefinite, and may either include only the earliest prodromata or the first symptoms after the fever has commenced, or both. The author understands the term "earliest stages" to include the period of incubation and the first seven days of fever, and has, therefore, endeavored to eliminate diseases which complicate the diagnosis after this time only.

The differential diagnosis of a disease presenting such multiformity of aspect as typhoid fever is necessarily difficult, and especially is this true in its earliest stages, when few infectious diseases exhibit such great variations in their manner of attack and onset. The extreme variability of the clinical factors to be considered, the intensity of the infection, the resistance of the patient, the sanitary surroundings, all render it impossible to tabulate with any degree of accuracy the differences existing between the earliest manifestations of typhoid fever and those of diseases which may resemble it. Were it possible to demonstrate with ease and certainty the presence of the bacillus typhosus or its products at every stage of the disease, these difficulties would cease to exist, but, unfortunately, no satisfactory method has yet been brought forward which fulfils all these requirements ideally.

During the period of incubation no entirely satisfactory bacteriological method of diagnosis exists, and even clinical signs are hazy and uncertain. Not only is it practically impossible to determine exactly the time of the receipt of the contagium, but it is equally difficult to mark the dividing line between the period of incubation and the actual commencement of the disease.

Different organs may bear the brunt of the infection and consequently the manifestations of the disease may be extremely variable; certain symptoms commonly present may be replaced or masked by others referable to the lungs, kidneys, or to derangement of the mental functions. As Herrick has so truthfully remarked, typhoid fever is not only

an imitator of other diseases, but many other diseases imitate typhoid fever. Even the length of the initial period is variable, occupying from one to three weeks, and at such times patients are rarely under observation.

The many forms which typhoid fever may assume also form an obstruction to early diagnosis. It may be abortive, mild, severe, hemorrhagic, renal, pneumonic, or ambulatory. It may be modified or masked by the strength of youth or the weakness of old age or childhood.

The ill-defined languor and indisposition, mental depression, headache, vertigo, sacral pain, anorexia, and irregularity of the bowels, in the absence of fever and physical findings, are in sharp contrast to the initial period of other febrile diseases, which during this stage generally present no manifestations. In those cases which come under close observation a careful examination of the temperature curve may show wide daily fluctuations of temperature, even though fever be absent. This taken into consideration with the symptoms will often put a careful clinician on the road to an early diagnosis. Pepper claimed to have been led to anticipate an attack of enteric fever by the unusual dulness of hearing and persistent occipital headache following a few days of malaise.

Epistaxis is of differential value simply as a part of the symptom-complex. It occurs, however, with far greater frequency in typhoid fever than in many other diseases, which are apt to be considered in the differential diagnosis. Its value is diminished chiefly by the fact that it also occurs early in cerebrospinal meningitis and in acute miliary tuberculosis.

The first febrile symptoms mark the actual commencement of the disease, the fever gradually increasing, and accompanied early by slight, oft-repeated chilly sensations. This occurs with such regularity that in cases ushered in by a hard chill and a sharp rise of temperature, typhoid fever is usually eliminated from the diagnosis at once. The temperature rises gradually with slight morning remissions and at the close of the first week usually reaches 40° C. The platted curve of these step-like gradations should be of great weight in the differential diagnosis. It should be mentioned in this connection, however, that while the gradually ascending temperature curve is typical of typhoid, there are cases occasionally in which there is a sharp initial rise in all respects similar to that of other infectious diseases.

Remittent malarial fever presents very irregular curves. Typhus, relapsing fever, scarlet fever, measles, and smallpox all exhibit a sharp initial rise of temperature. Intermittent malarial fever presents paroxysms of fever hardly to be mistaken for the steady progression of enteric fever. The disease presenting the febrile movement most closely resembling typhoid is Malta fever. If the history of exposure

be elicited, the diagnosis can only be made by the clinical and bacteriological findings. A differential diagnosis of this fever is almost impossible in the majority of cases without the aid of the microscope and the serum test.

It will also be observed that the pulse rate of typhoid fever, while it exhibits a striking parallelism to the temperature curve, is relatively infrequent and does not attain the rapidity which we are accustomed to find in like degrees of fever. In no other febrile disease does this occur with such regularity.

By the fourth or fifth day the pulse is already dicrotic and usually remains so throughout the disease. This occurs more often in typhoid fever than in all the other infectious diseases put together, and, as will be shown, is of great differential value.

Oddo and Audibert, in the *Gazette des Hôpitaux*, under the title "Le dicrotisme dans la fièvre typhoïde," give some interesting facts illustrative of the character of the pulse of typhoid. They speak especially of the frequency with which dicrotism occurs during the initial period, stating that in the great majority of their cases it was present on the day of entry of the patient into the hospital. In a few cases this phenomenon was an early symptom only, disappearing in a short time. The authors accordingly recognize several types, dicrotisme ephemere, and dicrotisme persistent, the latter being subdivided into dicrotisme continu, dicrotisme secondaire, and dicrotisme intermittent. In all, fifty cases were examined. Thirty-four of these presented dicrotism. Of these thirty-four, six were ephemeral and lasted from ten to forty-eight hours, and thirteen were continuous. In five cases the dicrotism was continuous, but disappeared about the twentieth day and reappeared about the twenty-fifth, and continued until the close of the disease. This is the so-called dicrotisme secondaire. In thirteen cases the dicrotism was intermittent, continuing for two or three days, with an intermission of about the same length of time. From these cases, which were not selected, it would appear that the dicrotism, though valuable, is a variable sign and should be carefully searched for, even though absent when the patient is first examined.

The average pulse tension of typhoid fever is about 140. Just what diagnostic import is to be attached to this fact the author is not prepared to state. From his limited experience with the tonometer of Riva Rocca, he is led to believe that careful tabulation of the pulse tension in various diseases, together with the accurate recording of the various factors which may tend to raise or to lower pulse tension, will prove of considerable value in differential diagnosis.

Of great diagnostic significance are the roseola, which appear during the latter half of the first week, and are distributed upon the abdomen, chest, and back. The roseolous exanthemata of other infectious diseases

in some cases resemble it, but rarely so closely as to deceive an experienced observer. Taken into consideration with the state of the spleen and the bowels, the time of the eruption and the subsidence of the rash, its succession of crops, its characteristic distribution and efflorescence, it is perhaps the most valuable single sign of the disease. Its value is enhanced by the fact that it is not preceded by an evanescent erythema as are most of the papular rashes of the acute exanthemata. Further, many reputable observers have recently determined that in the greater number of cases the blood obtained from the rose spots contains the bacillus typhosus.

The typhoid eruption appears later than that of any of the other exanthematous diseases; that of rötheln appearing on the first, scarlet fever on the second, measles and smallpox on the third, typhus on the fifth, and typhoid on the sixth or seventh day of the disease. The rash of rötheln is bright pinkish-red and lasts but two or three days; that of typhoid comes on in crops and the entire rash lasts much longer. Scarlet fever presents a subcuticular flush which may be so intense that the patient's skin may have the color of a boiled lobster, yet a noteworthy fact is the exaggeration of redness at certain points so that the skin has a mottled appearance. No rash like this occurs in typhoid. The eruption of measles is macular. The macules are dusky red and tend to coalesce and arrange themselves in crescentic areas. The typhoid roseola are papular, bright pinkish-red, and rarely coalesce. The pâpules of variola are shotty and do not disappear on pressure as do those of typhoid. Typhus presents a dusky subcuticular mottling. The typical typhoid eruption is raised above the level of the surrounding skin.

One of the most unique of recent diagnostic suggestions is that of Gibbes to recognize the rose spots by photographic processes before they are visible to the unaided eye. He uses an orthochromatic or non-halation plate, or, in the absence of one of these, a ray filter. Care is taken to focus exactly and the development is not pushed too far. A slow developer is used. By this means he has in several instances anticipated the roseola by several days.

In relation to the skin manifestations of typhoid, mention may be made of the drug rashes which may tend to cloud a diagnosis. Copaiba roseola are found most often on the extremities and do not possess the bright hue of those of typhoid. Furthermore, they appear suddenly, itch, and disappear on the withdrawal of the drug. The rash of quinine and atropine both resemble scarlet fever more closely than they do typhoid. Turpentine produces a blotchy rash, scarlatiniform in character, and only rarely resembling typhoid.

From the beginning of typhoid there is a progressive diminution in the number of the white blood corpuscles. There is usually also a

reduction in the number of the red blood corpuscles and a corresponding decrease in the percentage of hæmoglobin. The red cells rarely exhibit marked changes in form, size, or color. The latter changes are found in most infectious diseases, but the absence of leukocytosis is of significance in the elimination of pneumonia, cerebrospinal meningitis, sepsis, and other diseases usually accompanied with an increase of white corpuscles.

Although enlargement of the spleen is observed in all infectious diseases, it is of special diagnostic significance in typhoid fever. There are reported cases in which it is demonstrable during the period of incubation, but it is very unusual to find this condition before the middle of the first week. In few other infectious diseases does the enlargement occur so early or persist so long, if we except Hodgkin's disease and malaria. Exception must be made in the case of typhus, however, in which the enlargement occurs during the first days of fever.

The stools of typhoid fever present no chemical or physical diagnostic peculiarities, if the presence of the bacillus of Eberth be excepted. However, their thin liquid, ochre-yellow, "pea-soup" character, penetrating ammoniacal odor, tendency to separate into two layers, and their relative infrequency will always call to the mind of the clinician typhoid fever.

The diazo reaction of Ehrlich may be found first occurring in typhoid from the fifth to the thirteenth days, and continuing while the disease is at its height. It is diminished in value by the fact that it may be found at times in pneumonia, scarlet fever, malaria, variola, measles, septic conditions and advanced malignant disease. Its absence in a case which otherwise closely simulates typhoid decides rather against the latter.

The negative rôle of profuse sweating, herpetic eruptions, jaundice, coryza, conjunctivitis, and vomiting is to be noted. Profuse sweating would point rather to acute tuberculosis, relapsing fever, pyæmia, acute ulcerative endocarditis, or acute articular rheumatism than to typhoid. Herpes occurs with frequency in malaria, pneumonia, epidemic cerebrospinal meningitis, and after the ingestion of salicylic acid, but very rarely in typhoid fever. Jaundice would indicate Weil's disease, remittent malarial fever, or acute yellow atrophy of the liver, rather than typhoid. Coryza and conjunctivitis at the onset would cause the diagnostician to incline more to the belief that measles or influenza existed; and vomiting would point to variola, typhus, or cerebrospinal meningitis, rather than to typhoid. "It must be emphasized that the ordinary symptoms of coryza—sneezing, increased secretion, conjunctival catarrh—are among the greatest exceptions, at least in moderately severe and severe cases of typhoid fever, and may be thrown in

the balance against a diagnosis of typhoid fever. Severe infectious conditions with a predominating coryza generally have some other significance. Under such circumstances typhus fever and influenza especially would have to be considered."

Other symptoms referable to derangement of the respiratory organs have not uncommonly to be taken into diagnostic account. For the most part they obscure rather than aid in the diagnosis. Epistaxis has already been commented upon. It depends, as do most of the manifestations occurring in the upper air-passages in typhoid, upon the spongy hyperæmic condition of the mucous membrane. It is observed most frequently during the period of incubation and in the beginning of the febrile stage, and occurs in about seven per cent. of cases.

Tonsillitis and laryngitis occasionally occur early, and from the fact that the bacillus of Eberth has been repeatedly isolated from these locations, it would appear that in some cases, at least, the initial infection takes place in these organs. It may also be noted in this connection that the bacillus typhosus has been isolated from the sputum in the initial bronchitis sometimes met with. Just how much diagnostic weight is to be assigned to such findings it is hard to say, but with improved bacteriological methods it may be considerable.

The field which has long held forth the greatest promise of an infallible diagnostic method has been that of bacteriology. As has been stated, these expectations have in part to be fulfilled, but much has been already accomplished; the bacillus typhosus has been clearly differentiated from the colon bacillus; the specificity of the agglutinating action of immune serum has been demonstrated; cultures have repeatedly been made from the rose spots, urine, feces, sputum, and, what is of greater importance in the early diagnosis, from the blood itself.

Before considering the differences which exist between the bacillus typhosus and the colon bacillus, a brief description of the morphological and biological peculiarities of Eberth's bacillus will be à propos. The bacilli, as ordinarily seen, are short, thick rods about the length of one-third the diameter of a red blood corpuscle. The ends are rounded and their width is about one-third their length. They are actively motile and possess large numbers of flagellæ, which spring from the entire surface of the bacillus. In different environments the bacilli undergo various alterations in form, size, and arrangement. This has been the cause of many contradictory statements in regard to the biology of the organism. Undoubtedly there does exist a group of organisms which are intermediate in their biological manifestations between the typhoid and the colon bacilli. Whether these belong to the colon group or to the typhoid group or are in a separate division is a mere matter of nomenclature; the fact that they have been re-

peatedly isolated from the blood of patients, and the fact that these organisms display the Pfeiffer phenomenon with immune serum, is sufficient proof of their existence. They will be considered at greater length under the discussion of the elimination of paratyphoid fever from the diagnosis of typhoid fever.

The typhoid bacillus displays facultative anaërobiosis and grows readily upon the various culture media, especially the potato, at room temperature. The investigations of Gaffky into the cultural peculiarities upon the potato have been the nucleus from which have sprung many valuable methods for isolating the typhoid bacillus.

Of the special cultural and biological differences existing between the colon and the typhoid bacilli little need be said. The longer and more numerous flagellæ of the bacillus of Eberth, its greater motility, its cultural peculiarities on potatoes, litmus milk and glucose agar, and its specific reaction to the typhoid serum render its recognition comparatively easy.

The reaction of the colon bacilli to the serum of guinea-pigs immunized against it and the similar reaction of the paracolon group should be specially mentioned as differentiating them from the typhoid bacilli. To be sure, the typhoid bacillus also reacts to these sera, but only in the low dilutions, and therefore would not deceive a careful and experienced observer.

The Widal method of serum diagnosis has probably received more space in the typhoidiana of recent years than any other single sign of typhoid fever. As this paper includes the differential diagnosis of typhoid fever in its earliest stages only, any prolonged discussion of the methods and shortcomings of the agglutination test would be out of place.

The lysogenic action of the serum has proven in the hands of thousands of competent observers to be of inestimable value, but the statement of Widal that the reaction occurs as early as the end of the first week has been fulfilled only in the minority of cases. It is unfortunate for the early differential diagnosis that the occurrence of the reaction is usually delayed until the tenth day and may not present itself until late in the disease. It is equally unfortunate that the reaction occurs during health with the blood of individuals who have never had the disease; that it is found in the presence of other diseases, especially the acute infectious diseases; and that it sometimes persists for years after an attack of typhoid fever. It is to be remembered, however, that these errors are only apparent and that they occur only when the lower dilutions of the serum are used. With the higher dilutions and careful noting of the length of time elapsing before the reaction occurs, the liability to error will be greatly minimized.

Only very recently a method which combines the serum reaction of Widal and the culture of the bacilli from the stools, has been introduced

by Wolff, of Hartford. It is very original and evidently practical, as will be seen by the following excerpts from Wolff's original article:

"The technique is very simple. A loop from the feces of the suspected case is smeared upon the surface of an agar slant in a prepared tube. . . . . From this first specimen one or more bouillon cultures are prepared. The bouillon must react from 1 to 2 per cent. *alkaline* with the $\frac{n}{10}$ acid, using phenolphthalein as the indicator. The infected bouillon is now incubated at the usual temperature for twelve hours, when we are ready to make the examination. A sample of the blood is taken at the same time the specimen of the feces is procured. This is mixed with the bouillon culture by the usual procedure and placed upon the stage of the microscope. If now there is sufficient agglutinative material present, the typhoid bacilli (if they exist in the culture) will very shortly form clumps in the fields, which will be observed full of colon bacilli in active motion; and if this reaction does occur, we can of course safely say that the case has advanced at least to the second week of the disease. Should no reaction occur, another sample of the bouillon culture is tested with the blood from an advanced case of typhoid fever, the agglutinative power of which has been tested by the ordinary method, with a pure culture of the bacillus of Eberth. Indeed it is necessary for the proper use of this test to keep in stock a number of specimens of blood from well-marked typhoid cases. These can be kept in a dry place, and they retain their power to produce a reaction with a pure culture, probably indefinitely. With this blood, if the feces contain any typhoid bacilli, a positive and distinct reaction will shortly occur, the clumps of typhoid bacilli being more or less numerous according to the number of typhoid organisms present; while the still motile colon bacilli occupy the rest of the field, and are seen to be in active motion. This indicates that the case is one of typhoid fever, and that the disease is in an early stage, at least from the middle to the end of the first week. By means of this simple method we are enabled, 'in my opinion,' to make an accurate diagnosis in the early stages of the disease when the other symptoms may be more or less masked, and thus remove many elements of doubt in a suspicious case."

Within the last two years blood cultural methods have been constantly brought forward which demonstrated the bacilli in the general circulation. Recently, however, the bacilli have been demonstrated prior to the occurrence of the Widal reaction and, in a few instances, before the enlargement of the spleen or the appearance of the roseola.

The method of Seeman for examining the blood obtained from the rose spots is perhaps the simplest. A drop of bouillon is placed over the sterilized skin and an incision made into the rose spot through the bouillon. A little of the blood from the rose spot is then squeezed out and mixed with the bouillon, which is examined by the ordinary cultural methods.

The examination of the blood obtained from the rose spots is not without limitations, especially in the early diagnosis. The spots do not

appear before the sixth or seventh day as a rule, and in doubtful cases may be absent even then. To be of value the rose spots must be quite fresh, as the old bacilli are too weakened to grow well on ordinary media. Further, if the observer is competent to make a bacteriological examination of the fluid obtained from a rose spot, he is just as competent to examine blood obtained by venesection.

The technique of this method of investigation is comparatively simple and can be easily carried out in any fairly well equipped laboratory. The most important factor is the avoidance of contamination. To this end, the arm at the bend of the elbow is prepared by the usual surgical methods and a wet bichloride dressing allowed to remain until the time of taking the blood. The hands of the operator are prepared as for an aseptic operation. A constriction is placed above the patient's elbow to distend the veins and the skin is anæsthetized by pure carbolic acid or ethyl chloride. A solid metal or glass syringe, provided with a tight piston and a sharp needle, is boiled five minutes. The needle is passed directly into (not through) the vein, great care being taken not to contaminate it in any way during the procedure. Five cubic centimetres of blood are withdrawn and distributed equally between five Erlenmeyer flasks, each containing 100 c.c. of bouillon. These are well shaken to distribute the bacilli from within the clots, and incubated for twenty-four hours, when if a culture is apparent transplantations may be made on the various media. The original flasks may be incubated twenty-four hours longer and a stab culture then made on agar. If the hanging drop at this time shows a motile organism, a six-hour culture in bouillon is made and the Widal reaction tried with a known immune serum. The results of this method have proven very satisfactory, and its value in early diagnosis is shown by the fact that it occurs in about 87.5 per cent. of cases during the first week.

As an evidence of the high diagnostic value of blood cultural methods in diagnosing typhoid, may be quoted Schottmüller, who in his series of 119 cases discovered the bacillus of Eberth in the blood in 84 per cent. The earliest case in which it was possible to make an examination was on the second day of the disease, and the result was positive. In a great many cases typhoid bacilli were discovered before the Widal reaction occurred. These investigations must naturally alter our idea of the pathology of typhoid, in that they show that throughout the entire course of the disease the bacilli circulate in considerable number in the blood. This accounts for the occurrence of the roseola and remote inflammatory changes during the course of the disease.

Typhoid bacilli have been frequently isolated from blood obtained by splenic puncture. The date of their appearance in splenic blood is probably very early, and, from a diagnostic standpoint, very important. This has, in the past, led many "who rush in where angels fear to

tread" to adopt this method of procedure, regardless of the very grave consequences which it may entail. The practice is now discountenanced by most investigators and has given way to the examination of the blood obtained from other sources.

Typhoid bacilli have long been known to exist in the stools and have been isolated in many cases, but only after the use of much difficult laboratory technique. The great objection to the bacteriological examination of the stools has been the extreme difficulty in separating the typhoid bacilli in pure culture uncontaminated with the colon bacillus.

The method of Remy has proven successful in the hands of several investigators. The principle underlying it is the use of a medium which endeavors to approximate the chemical constitution of the potato, and to this end definite amounts of asparagin and several of the inorganic salts are added. Just before using, a little milk-sugar and two drops of 1:40 solution of carbolic acid are added to each test tube. The feces are diluted about 1:8000 and plated with the melted medium. At room temperature colonies appear in forty-eight hours. The colonies are transplanted on bouillon and examined for motility and also cultivated to determine gas or indol production. By this method the bacilli have been found in several reported cases before the occurrence of the Widal reaction.

Several other equally good methods, each working on a different principle, have been introduced within the past two years. That of Hiss has met with considerable approval, and, as he has outlined his method with far more clearness than the author can, a portion of his original article on the subject is herewith inserted:

"Two media are used: one, for the differentiation of the colonies of the typhoid bacilli from those of the colon group, by plate culture; and one, for the differentiation of these forms in pure culture, in tubes.

"The plating medium is composed of 10 gm. of agar, 25 gm. of gelatin, 5 gm. of sodium chloride, 5 gm. of Liebig's extract of beef, 10 gm. of glucose, and 1000 c.c. of distilled water. The final titration of this medium should indicate the presence of about 2 per cent. of normal acid (1.8 per cent. to be exact), phenolphthalein being the indicator; and the medium should be brought to this acidity by the addition of normal hydrochloric acid solution.

"The growth of the typhoid bacilli in plates made from this medium gives rise to small light greenish colonies with irregular outgrowths and fringing threads. The colon colonies, on the other hand, are much larger, and as a rule are darker and do not form threads. This medium is practically solid and the differentiation seems to depend upon the fact that typhoid bacilli form threads in a medium of this acidity when peptone is absent.

"The tube medium contains 5 gm. of agar, 80 gm. of gelatin, 5 gm. of sodium chloride, 5 gm. of Liebig's extract of beef, 10 gm. of glucose,

and 1000 c.c. of distilled water, and should react 1.5 per cent. acid, phenolphthalein being the indicator.

"In this semisolid medium the growth of the typhoid bacillus produces uniform turbidity at 37° C. within eighteen hours. The colon cultures do not give the uniform clouding and present several appearances, dependent upon differences in the degree of their motility, and upon their power to produce gas in the medium.

"The usual method of making the test is to take enough of the specimen of feces—that is, from one to several loopfuls—and transfer it to a tube containing broth, making the broth fairly cloudy. From this emulsion five or six plates are usually made by transferring one to five loopfuls of the emulsion to tubes containing the melted plate medium, and then pouring the contents of these tubes into Petri dishes. These dishes, after the medium has hardened, are placed in an incubator at 37° C. and allowed to remain for eighteen to twenty-four hours, when they are ready for examination. If typical colonies with fringing threads and outgrowths are found, the tube medium is inoculated from them and placed in the incubator at 37° C. for eighteen hours. If these tubes then present the characteristic clouding, our experience indicates that the diagnosis of typhoid may safely be made; for the bacillus of typhoid alone, of all the organisms occurring in feces investigated during these experiments, has displayed the power of giving rise both to colonies with fringing threads in the plating medium, and the uniform clouding in the tube medium, when exposed to a temperature of 37° C.

"A diagnosis may thus be made in thirty-four to forty-eight hours. If doubt is entertained as to the distinctiveness or value of these characters, the bacillus may be further tested against a dilution of typhoid serum."

Higley believes that the method of Hiss has given slightly better results in his hands than the Widal reaction. It has occurred in a few instances earlier by several days. The method is more difficult than the cultural examination of the blood. It remains to be seen if it is in any way superior to it, but this does not detract from its value as a link in the chain of diagnostic evidence.

It is doubtful if the bacillus typhosus occurs in the urine sufficiently early to be of material aid in the early diagnosis. In a few isolated cases they have been found as early as the sixth day, but this is very rare. Later they may occur in such enormous numbers as to cause a peculiar shimmer when the urine is shaken.

What has been said of the urine applies even more forcibly to the expectoration, sweat, and expired air. That the first may contain the bacillus of Eberth early in the disease is not to be denied; but this occurs most often in typhopneumonia, which is a rare early complication. The elimination of the bacilli in the sweat, tears, and expired air is too infrequent to entitle them to diagnostic importance.

The method of Moore acts upon the combined principles of the lysogenic action of the colon serum and the motility of the typhoid organism. In one arm of a W-shaped tube containing bouillon, to

which has been added the serum obtained from a rabbit immunized to the colon bacillus, is planted a loopful of the culture from which it is desired to obtain the bacillus typhosus uncontaminated. The serum causes the Gruber reaction to occur with the colon bacilli and they are agglutinated and precipitated. The typhoid bacilli emigrate and may be obtained in pure culture in the other arm of the tube.

Still another method is that of Biffi, who also utilizes the agglutinating serum of the bacterium coli. Contrary to Cambier, he found that the colon bacillus would pass through an earthen filter quite as readily as the bacillus typhosus. He accordingly first introduces the substance to be examined into bouillon, to which has been added a serum which will agglutinate all the varieties of the colon bacilli. This is prepared according to the method of Pfeiffer for preparing typhoid agglutinating serum. The rabbit which is to furnish the serum should be injected with all the varieties of the colon bacilli, so that he shall be equally immune to all the varieties, and his serum able to agglutinate any species of the colon bacilli which may be present in the substance to be examined. This serum should be tested as to its agglutinating power and a quantity added to the bouillon proportional to its agglutinating ability.

The foregoing has considered the general data for diagnosis and has indicated their application in a few instances. The specific differences which exist between typhoid fever and the diseases which may resemble it must now be considered.

During the developmental period of the acute exanthemata they may present symptoms which will render difficult the differential diagnosis. This is especially true of measles, scarlet fever, variola, and typhus. The knowledge of exposure to any one of these diseases will of course aid materially, but the most reliance can be placed upon the initial symptoms. Perhaps the earliest manifestation of all the eruptive diseases occurs in the pharyngeal mucous membrane; such involvement is very rare in typhoid fever. The coryza and conjunctivitis of measles, the angina of scarlet fever, and the initial backache of smallpox are all in contradistinction to the onset of typhoid. Variola presents in addition an initial rash, which may be as diffuse and vivid as a true scarlatina. A careful observation of the wrists and hair line for shotty papules will usually prevent error.

Confounded and associated under the same name until the middle of the nineteenth century, it is not surprising that, even at the present time, some difficulty may exist in making an early and accurate diagnosis between typhus and typhoid fevers. Prior to the appearance of the eruption it may be almost impossible. The uneventful period of incubation, followed by a chill and an abrupt and rapid rise of temperature; the extreme rapidity of the pulse; the early vomiting and extreme

prostration of typhus are in sharp contrast to the step-like gradations of temperature, slowness of the pulse, and absence of early vomiting and prostration of typhoid fever. Proportional to the rapid rise and severity of the fever are the profound disturbances referable to the derangement of the nervous system which occur earlier and with greater severity than in typhoid.

Not infrequently typhoid patients continue on duty during the first ten days of the disease, and delirium and coma do not occur until late. In typhus, on the other hand, they are prostrated at the very onset of the disease, and delirium, stupor, and coma may rapidly succeed one another.

The blood findings of the two diseases present marked differences; typhus showing a moderate leukocytosis, while in typhoid an actual diminution of the white cells occurs. Typhoid presents in the great majority of cases the specific bacillus before the expiration of the first seven days. Thus far no organisms have been found in the blood of typhus patients. Later typhoid blood shows the Widal reaction, which does not occur with typhus serum.

Far more important and readier of demonstration are the skin eruptions of the two diseases. That of typhus occurs earlier, and in typical cases presents such marked differences that the differentiation is easy for an observer of experience. It should be mentioned in this connection, however, that typhus cases do occur in which it is very imperfectly developed or entirely absent. The exanthem of typhus is distributed with uniformity over the trunk and limbs. It is neither well defined nor sharply limited. It is macular, hemorrhagic, and distinctly petechial. It appears in a single crop, a second eruption being practically unknown. It has a dusky red, coppery hue, and appears as if beneath the surface of the skin. The eruption of typhoid, on the other hand, usually involves the trunk alone. It is sharply defined, papular, and purely hyperæmic. It appears in crops, is bright pinkish-red and is slightly elevated above the surface of the skin.

The face of typhoid fever early exhibits bright eyes and slightly flushed cheeks, later a dull and apathetic countenance. Typhus, on the contrary, presents a swollen, livid-red appearance, with injected conjunctivæ, contracted pupils, and an agitated expression.

The disease which may occasion the greatest difficulty in early differentiation is acute miliary tuberculosis. Many cases are under observation for weeks before the diagnostician can arrive at a conclusion, and then perhaps only after the recovery or death of the patient. Both have this in common, that the manifestations of each are due to the action of similarly acting toxins. The initial malaise, headache, anorexia, and irregularity of the bowels is present in both. Each presents enlargement of the spleen, but it occurs earlier and more markedly in

typhoid. Both present the Ehrlich diazo reaction and febrile albuminuria. The reddish spots which occur on the abdomen in miliary tuberculosis may cause confusion. They do not appear in crops and are much less abundant than the roseola of typhoid. Profuse sweating occurs much more often in miliary tuberculosis. An important difference occurs in the temperature pulse curve of the two diseases, the marked irregularity of temperature, with a proportionally rapid pulse, of acute phthisis being quite the opposite of the steady ascent and comparatively slow pulse of typhoid. Acute phthisis presents Kernig's sign; typhoid never. Dicrotism is rare in miliary tuberculosis. There may be, though unusually, a leukocytosis in acute tuberculosis, and the bacillus tuberculosis has been found in the blood of a few cases. The absence of leukocytosis would not necessarily decide in favor of typhoid, but the discovery of the bacillus of Eberth in the blood, or the occurrence of the Widal reaction, would. The relative increase in the large mononuclear leukocytes found in typhoid does not occur in acute tuberculosis. In the minority of cases the eye-grounds show choroidal tubercles. This is a decisive condition when present. Tubercle bacilli are rarely found in the sputum of an acute tuberculosis, and the lung findings may be exactly the same at the beginning of both diseases. There is, however, a greater tendency to respiratory frequency and slight cyanosis in miliary tuberculosis. Curschmann considers acute pulmonary emphysema "an especially decisive objective sign" never occurring "as the result of typhoid bronchitis."

In peritoneal tuberculosis the persistent abdominal pain and physical signs of effusion will make the diagnosis. Very early a decision will rest upon the physical findings indicative of tuberculosis of other organs.

When tubercular meningitis accompanies the general process, the diagnosis is rendered somewhat easier. The sudden onset with a convulsion, or severe headache and high fever; the agonizing pain; projectile vomiting; hydrocephalic cry, and contracted pupils go to make up a picture widely different from that of typhoid. The pulse of basilar meningitis is at first small and rapid. Subsequently it is as slow as in typhoid, but is irregular and rarely dicrotic. Quincke's lumbar puncture should never be omitted in doubtful cases. If the tubercle bacilli be present they will be discovered on centrifugalization of the spinal fluid, and will, of course, determine the diagnosis.

Cerebrospinal meningitis, however, is not so readily differentiated from those cases of typhoid ushered in by headache, photophobia, delirium, retraction of the head, twitching of the muscles, and even convulsions. It is easy to make a decision when an epidemic of one or the other is prevailing, but it is in sporadic cases that the chief difficulty lies. The irregular and variable temperature, the marked

increase in the polynuclear leukocytes, and the profound psychical disturbances have no great resemblance to typhoid. The cutaneous symptoms of the two diseases are very different. Herpes occurs with great frequency in cerebrospinal meningitis, but almost never in typhoid. The rash of the first is petechial and is sometimes distributed over the entire skin. That of the latter is hyperæmic and usually limited to the trunk.

The examination of the fluid obtained by lumbar puncture for the diplococcus intracellularis meningitidis of Weichselbaum is the most reliable method of diagnosis. Blood cultures should also be made to determine the presence of the bacillus of Eberth, and with the newer bacteriological methods will prove of great value.

Meningitis or cerebral abscess from ear disease may sometimes resemble typhoid. The history of sudden cessation of a chronic ear discharge, followed by a rise of temperature, nausea, vomiting, and the symptoms of an acute septic infection engrafted on an existing chronic saprogenic suppuration, would certainly point to a purulent meningitis. The careful examination of the mastoid will sometimes render a decision.

Irregular forms of malarial fever, particularly when due to infection with the æstivo-autumnal parasite, may closely resemble typhoid fever. The onset of typhoid differs from that of remittent fever, in that that of the former is gradual and progressive, with slight chilly sensations and step-like gradations of temperature which rarely reach 40° C. before the fourth day; while the onset of remittent fever is generally intermittent, with severe chills and irregular remissions of temperature which may reach 40° C. in twenty-four hours or less. The temperature of malarial fever disappears under the use of quinine, while that of typhoid is not influenced by it. The grayish color of the face, the subicteric sclera, and the anxious, restless expression of remittent fever are all quite the opposite of the facies of typhoid, which early presents flushed cheeks, clear sclera, and an alert but not anxious countenance. Herpes is common in æstivo-autumnal fever, but rare in typhoid. Early delirium is rare in typhoid, but when occurring is persistent and variable only in degree. The delirium of remittent fever, on the contrary, may come on in the early days, is recurrent, and changes with the exacerbations of temperature and other symptoms.

An increase of the lymphocytes to 40 per cent. or over, without any increase in the large mononuclears, points to typhoid as against malarial fever. An increase in the large mononuclears to 12 per cent. or upward, especially during the remissions of temperature, indicates malaria rather than typhoid. The presence of myelocytes in any such number as from 1 to 5 per cent. indicates malaria rather than typhoid. A high degree of anæmia is more common in malaria. A very great reduction in the total leukocyte count is more frequently met with in malaria

than in typhoid fever; while the proportion of white to red corpuscles in malaria is not infrequently less than 1 to 2000, which is rare in typhoid fever.

Finally, it is to be noted that cases have been reported in which typhoid fever is superimposed upon a malarial infection; and that, in these cases, the blood not only contains the bodies of Laveran, but also the typhoid bacillus. Fortunately such an occurrence is rare, at least it is rarely recognized; but the knowledge that such double infections do occur will sometimes call for more careful clinical and bacteriological examination.

Remittent fever presents no typical exanthem and the urticaria, which occurs not uncommonly, is very different from the roseola of typhoid. The early anæmia of æstivo-autumnal fever is not found in typhoid. The blood in the former shows leukocytosis, without diminution in the eosinophiles; that of the latter, no leukocytosis and marked diminution of the eosinophiles. Further, typhoid blood shows no malarial parasites or pigmented leukocytes, but, on the contrary, the typhoid bacilli and the Widal reaction.

Plague may occasionally be mistaken for typhoid fever, but the reverse will very rarely happen. The history of exposure to plague may be obtainable, though not commonly. The period of incubation of plague is much shorter than that of typhoid, nine days being the extreme limit. The prodromes may be entirely absent or at least of such mild character as to be unnoticed by the patient himself. Occasionally they may exactly duplicate those of typhoid, but pain and stiffness in the joints and tenderness in the groins or axillæ will be present also in the glandular type. The stage of invasion with chills, rigors, or sensations of heat is in sharp contrast to typhoid fever. In pestis siderans, the overwhelmingly sudden onset, with rapidly succeeding delirium, vomiting, hæmatemesis, hæmaturia, melæna, coma, collapse, and death, has no parallel in typhoid fever. The presence of the bacillus pestis in the blood and the reaction of the serum to Pfeiffer's phenomenon are final distinguishing points from typhoid.

Influenza of the gastrointestinal form may be readily mistaken for typhoid. The abrupt onset, early prostration, and multiplicity of symptoms found in typical cases of influenza are very different from the gradual onset of typhoid. The absence of splenic enlargement and the typical roseola in influenza should also be noted. In those typhoid cases in which the nervous element preponderates, examination of the blood may be necessary to make a diagnosis. The presence of an epidemic, the contagious nature of the affection, and the presence of Pfeiffer's organism all point to influenza.

Cases of typhoid presenting marked pulmonary symptoms at the onset may be readily confounded with lobar pneumonia. On the other

hand, cases of pneumonia with insidious onset may be mistaken for typhoid fever. This is particularly true of the so-called senile pneumonias, and also of those cases in which the pneumonic process commences in the centre of the lung. Osler says, "Nervous symptoms are more frequent in pneumonia than in typhoid, and from the onset may so dominate that the local lesion is entirely overlooked." The absence of leukocytosis in typhoid and the presence of Eberth's bacillus in the blood and the dejections are of great differential value. The presence of the Widal reaction will, of course, be decisive. If the bacillus typhosus be found, the case may be considered pneumotyphoid. It should be mentioned in this connection that there occur, not infrequently, cases in which a diplococcus pneumoniæ is engrafted upon typhoid. It is not to these cases that the term pneumotyphoid is applied, but to those whose manifestations depend upon the bacillus of Eberth alone.

Pyæmia and other septic processes may sometimes require differentiation from typhoid fever. It is in such cases that the examination of the blood, with the view of determining the presence of Eberth's bacillus and the Widal reaction, and the absence of leukocytosis in typhoid, will prove of great value. In differentiating typhoid from puerperal septicæmia, the fact that pregnant women ill of typhoid usually abort may sometimes prevent error. The appearance of the roseola and the serum reaction will end all doubt in the matter.

Another pyæmic process which may occasion great difficulty is malignant endocarditis. Both diseases present enlargement of the spleen, abdominal tenderness, and diarrhœa; each shows delirium, stupor, and progressive exhaustion. If the heart was previously intact, symptoms referable to a cardiac lesion would be almost pathognomonic, as ulcerative endocarditis complicating typhoid usually occurs very late in the disease, at a time when the diagnosis has already been made. The temperature of ulcerative endocarditis is less regular in type than typhoid, and chills and sweats are far more common. Leukocytosis is marked in malignant endocarditis, but is absent in typhoid unless inflammatory complications occur. Furthermore, cardiac distress occurring in the course of typhoid is usually devoid of the extreme oppression and shortness of breath of endocarditis.

Infectious osteomyelitis, the "typhe epiphysaire" of Chassaignac, may simulate typhoid. The examination of the epiphyseal regions of the long medullated bones, and the inspection of the extremities for œdema, livid redness, and points of circumscribed tenderness will yield valuable information. Another diagnostic point is the presence of leukocytosis and the absence of the serum reaction and the specific bacilli from the blood in osteomyelitis.

As has been previously pointed out, Malta fever may very closely resemble typhoid. The temperature curves of the two diseases may be

almost identical, and both give rise to headache, insomnia, and anorexia. The presence of the micrococcus melitensis in the blood, and the serum reaction with this germ would be diagnostic of Malta fever. Probably the micrococcus is present in all the dejections. It has been isolated from the urine and blood repeatedly. Sweating and violent joint pain are common and early symptoms in nearly all cases of Malta fever. Joint pain may occur early in typhoid also, but rarely with such severity as mentioned above.

Relapsing fever, at the onset or at the beginning of an epidemic, may be mistaken for an anomalous typhoid. The temperature of febris recurrens nearly always rises suddenly at the onset and remains in the neighborhood of 40° C. from three to seven days, when it suddenly falls by crisis. High initial temperatures are rare in typhoid, and a fall by crisis practically unknown. The presence of the spirillum of Obermeier in the blood of relapsing fever, and the bacillus of Eberth in that of typhoid, together with the rarity of relapsing fever, all aid in the diagnosis.

There are times when trichiniasis with predominant gastroenteric symptoms may closely simulate typhoid fever. Distinguishing points are the presence of vomiting, œdema of the face and eyelids, and extreme myositis in trichiniasis.

Trichiniasis rarely presents the characteristic typhoid roseola or the enlargement of the spleen. The flexor contractures of the arms and legs, the painful swelling and tension of the muscles, the profuse sweating and itching of the skin, all make a much different picture from typhoid. The marked leukocytosis, especially the extraordinary increase in the eosinophiles, is in strong contrast to typhoid. The examination of a portion of the pectoral muscles will render final decision.

"Trichinosis and typhoid fever have been frequently associated, but most commonly the trichinosis has been in the patient and the typhoid fever in the mind of the physician. The association in the patient of these two diseases appears to be exceedingly rare." There are only two reported cases to be found in the literature of the subject, and this extreme rarity would almost exclude such a condition from the diagnosis, but might, in some cases, require an examination both of the blood and the muscle section.

Weil's disease with marked gastrointestinal symptoms may sometimes simulate typhoid. Its mode of onset, the history of exposure to cold, and the fact that it occurs most often in brewers, butchers, and ice-plant laborers, are all diagnostic points. The jaundice is severe and early. As has been before pointed out, this occurs but rarely in typhoid. The temperature is high and remains so from the beginning of the disease. There is usually enlargement of the liver and subcutaneous œdema over the hepatic area. The blood may contain the

bacillus proteus fluorescens, which is in marked contrast to the bacillus of Eberth.

Very mild cases of typhoid are apt to be diagnosed simple continued fever, in the early stages especially. The examination of the blood for Eberth's bacillus and the Widal reaction will settle the diagnosis.

During an epidemic of typhoid fever, catarrhal enteritis, especially in children, may give rise to symptoms like a mild or abortive attack of enteric fever. The absence of splenic enlargement, the rose spots, and the Widal reaction will usually determine the disease.

Papular syphilides may resemble the typhoid roseola. Usually they are easily differentiated, but Curschmann speaks of a case in which not only the eruption but also the general symptoms closely simulated typhoid. On the whole, a papular syphilide is of a darker and more coppery hue and more generally distributed. In these cases the history of a preceding initial lesion may aid materially.

Acute glanders with marked gastrointestinal symptoms may be suggestive of typhoid. However, the characteristic rash and the presence of the bacillus mallei will make a diagnosis. The use of mallein for diagnostic purposes is also of value.

Cases of prolonged appendicitis with slow onset will present differences which require careful investigation to be discovered. The pulse will be higher in proportion to the fever, and there will be much smaller remissions of temperature, and rarely roseola or the diazo reaction. There is marked leukocytosis.

Weiss, in his admirable paper read before the American Medical Association at the Chicago meeting in 1900, detailed a method for the staining of blood to determine the presence of suppuration in the body. The stain he used is as follows:

℞.—Iodi sublim.	1
Kali iodati	3
Aqua destil.	200
Gummi arab. q. s. ad consistentiam syruposam.	

A drop of blood taken from the lobe of the ear is carefully pressed between two cover-glasses so as to get as thin a smear as possible. This is air dried or fixed, after which a drop of the staining solution is then added to the slide, and the specimen is then ready for examination with the microscope. Blood from a perfectly healthy individual shows a dark yellowish staining of the red corpuscles. The nuclei of the white corpuscles take on a lemon-yellow colored, very glossy appearance, while the body of the cell is a slightly darker yellow. Normal blood also contains brown granules (extracellular glycogen).

The blood gives an altogether different reaction if suppuration be present. There is a great increase of extracellular glycogen, as shown by the large numbers of dark-brown granules present in the stained

blood. The leukocytes assume a brownish hue, varying in intensity from reddish-brown to dark yellow. The polynuclear neutrophiles are almost exclusively concerned in this reaction, as it never appears in the eosinophiles.

The value of this method in eliminating suppurative appendicitis from the diagnosis is great. It is easy of application and in the hands of the author has proven very satisfactory.

An affection whose clinical manifestations may be identical with those of typhoid is paratyphoid fever. Since 1896, when Archard and Bensaude made their first report, eighty-four cases have been recorded; and, without doubt, many, which hitherto have passed as true typhoid, but which have presented no Widal reaction, except in low dilution, are to be classed under this head. The symptoms during the period of incubation are in most respects identical in the two diseases, headache, malaise, anorexia, irregularity of the bowels, rose spots, enlargement of the spleen, and a gradually ascending temperature being the rule. Epistaxis has been noted in a number of instances, and the diazo-reaction of Ehrlich may be given by the urine. In uncomplicated cases the blood shows no leukocytosis. On the whole, the paratyphoid is milder and rather shorter than true typhoid; but in the early stages a differential diagnosis may be almost impossible. In low dilutions of the serum, even the Widal reaction occurs positively.

The knowledge which we now possess in regard to this disease has been the result largely of the practice of making blood cultures, and this still remains the surest method of diagnosing the disease. Two species of paratyphoid bacilli are recognized. Buxton classifies them as the alpha and beta paratyphoids. The alpha produces less gas in glucose media and resembles the typhoid in its action on milk. It differs from the beta paratyphoid in that the acidity on litmus milk is persistent, while that of the beta paratyphoid is finally changed to alkalinity.

Both of the paratyphoid organisms present Pfeiffer's phenomenon with immune serum. As Pratt has clearly pointed out, the blood should be tested with both species of the paratyphoid bacilli whenever there is any doubt about the diagnosis. As an example of this he quotes a case in which the serum gave a negative reaction with the alpha paratyphoid in 1:10 dilution, but completely clumped the beta paratyphoid in as high a dilution as 1:500. There may possibly be cases in which typhoid and paratyphoid coexist. At least it would seem so from some of the recent bacteriological findings. Pratt cites an instance in which "Bain, working in Dr. F. C. Shattuck's wards at the Massachusetts Hospital, found a case of typhoid fever the blood of which agglutinated the bacillus paratyphoid immediately and completely in dilution of 1:10. In higher dilution up to 1:200 there was clump-

ing without loss of motility. There was no reaction with the typhoid bacillus in dilution of 1:10. A culture from the blood, however, yielded a pure, abundant growth of the bacillus typhosus. The case died. Unfortunately no autopsy was held." Everything taken into consideration, the surest method of diagnosing paratyphoid from true typhoid lies in the cultivation of the bacillus from the blood of the suspected case.

There are times when the medical officer is called upon to make what might be called "a sanitary diagnosis," and to determine the presence or absence of typhoid fever.

The writer refers to those cases in which epidemics of disease occur—*e. g.*, during the Spanish-American war; and boards of officers are called upon to decide as to the nature of the infection. Under such circumstances they would be required to make the diagnosis of the disease in its earliest stages but very rarely; as in the great majority of instances cases in all stages could be seen, and by the averaging of the signs and symptoms in the various cases, and making close investigation into the food and water supplies, and the methods employed in the disposal of refuse, a diagnosis could be accurately arrived at.

CONCLUSIONS. 1. There is no single symptom on which alone an early diagnosis of typhoid fever can be made. It is only by careful consideration of the symptom-complex that a clinical diagnosis can be arrived at.

2. The most trustworthy, as well as the earliest, sign of typhoid fever is the presence in the circulating blood of the bacillus of Eberth.

3. The demonstration of the bacillus of Eberth in the blood is not beyond any fairly well equipped laboratory.

4. The bacillus of Eberth is found in the feces later than in the blood, but with comparative ease. The presence of the bacillus typhosus in the feces is of great value as a corroborative sign.

5. The presence of the bacillus typhosus in the rose spots is a trustworthy sign, but has no advantages over the examination of the blood from other localities.

6. The serum reaction of Widal is seldom demonstrable during the earliest stages of typhoid fever. It is of value only in the higher dilutions.

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THE LOCALIZING DIAGNOSTIC SIGNIFICANCE OF SO-CALLED HEMIANOPIC HALLUCINATIONS, WITH REMARKS ON BITEMPORAL SCINTILLATING SCOTOMATA.

BY PROFESSOR A. PICK,
OF PRAG.

A SHORT time ago Jolly[1] proved that a form of excitation phenomenon in the hemianopic field of vision, viz., scotoma scintillans, has not its origin in the cerebrum, especially not in the cortex, but in the primary optical tracts. I concur with this observation and shall verify the same absolutely in the following: My aim, however, is to prove that more complicated phenomena than elementary light sensations, as characterized by the scintillating scotoma—*i. e.*, real hallucinations—may be produced in the hemianopic field of vision by localized focal or functional affections in the optic tracts. The commonly accepted idea, therefore, that such hallucinations are of value for the localization of the lesion in the occipital lobe, is not correct. The cases at my disposal, for the most part from private practice, have not been verified by autopsy, but the conclusions to be derived therefrom as to the site of the lesion are so clear, that even a hyperskeptical critic should have no doubt. This theory has already been exploited by Uhthoff,[2] by means of his own observations, by what he found in

[1] Berliner klinische Wochenschrift, 1902, Nos. 42–43.

[2] Monatschrift f. Psych. and Neurol., 1889, Bd. v. I must here remark that American authors, especially Seguin and Peterson, deserve a great deal of credit in this question.

literature, and also by the report of a case with autopsy; but the force of his conclusions is restricted by his own doubts, and perhaps this is the reason that the original theory still holds good. On this account it will not be superfluous to bring new material to clear up this question. On the subject of diagnosis, as to the site of lesion, I shall be as brief as possible.

CASE I.—Bricklayer, aged sixty-two years, came into the clinic January, 1902. Had a purulent discharge from the ear as a child; since then hearing impaired on left side; had trachoma while a soldier, otherwise healthy. Suffers from trigeminal neuralgia since winter before last; impaired vision of five years' duration.

Six months ago he had a slight apoplectic seizure, a sudden feeling of weakness, causing him to drop an article, which he held in his hand, and fall to the floor. He was stunned but not unconscious; heard what was spoken, but could not understand all that was said. Spoke irrationally, disconnectedly, and knew that he did so, but could not help it. This condition lasted for from three to four hours, then he fell asleep. On awakening he felt much better, spoke again rationally and the general weakness gradually subsided. About a fortnight later he had a second attack, which took the same course as the first; only the patient observed, immediately after he was picked up from the floor, that the right half of the field of vision of both eyes had disappeared. These right halves of the visual field were represented by a black shadow divided from the left seeing halves by an exactly vertical line. This shadow slowly became paler without changing its form. Two weeks later he had another attack, the shadow moved still further into the field of vision, but returned to its former limit after a few hours. Three times more he had attacks, accompanied by the same phenomena—*i. e.*, general weakness, a faulty understanding of what was said to him, and an irresistible impulse to irrational, disconnected speech. Later he had a fourth, but lighter attack.

The somatic examination showed atheroma, vision reduced to 0.5 on both sides due to a commencing cataract, fundus normal, right homonymous hemianopsia, fields of vision as shown in Fig. 1.

Extrinsic ocular muscles normal, as was also the pupillary reaction. In the left eye accommodation was almost *nil*. No painful pressure points in course of left fifth nerve. The right corner of the mouth a little the lower, right palpebral aperture somewhat wider than the left; no other disturbances of motion except a slight paresis of the right leg; sensation normal; knee-jerks on both sides considerably increased. No Babinski reflex. Closer questioning shows conclusively that the disturbances of speech and writing following an attack were paraphasia and paragraphia. The patient himself cites the example that instead of saying "close the door," he said "close the closet," knowing full well that he spoke incorrectly. Even now symptoms of paragraphia can occasionally be demonstrated.

The patient, who calls at the clinic from time to time, told the following story January 15, 1903: About six months ago, while walking, he suddenly observed that everything on the right side of his field of vision was of a reddish color, changing later to green. Then he noticed for about half an hour at his right side a dog walking along with him,

the dog always appearing on the side on which he usually saw nothing. It looked like a real dog, but he convinced himself that it was only a shadow. Another time a girl with a colored shawl on her head walked

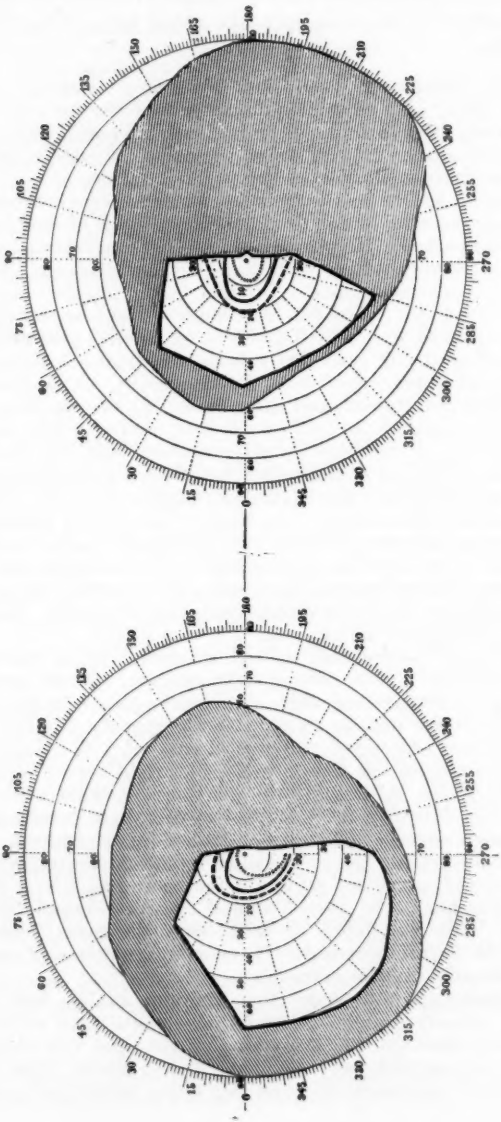

FIG. 1.

on his right side, but again he convinced himself that it was only a phantom. The color also was delusive. In general, he reports that occasionally he has slight attacks; as, for instance, on this day he had a feeling of chilliness and numbness in his left leg. Examination shows deviation of the tongue toward the right side, weakness of right arm, sensibility normal. It must still be remarked that the patient typically exhibited for a long time the faulty halving of horizontal lines, which phenomenon was first studied by Liepmann.[1] This symptom disappeared later, so that the patient now halves the lines correctly.

The phenomena described in the foregoing case and which presented themselves at first give rise to the assumption that we here have to deal with a focus of softening in the region of the left gyrus angularis, especially participated in by its medullary mass. The initial phenomena of paraphasia and paragraphia can be interpreted as neighboring symptoms, while the permanent hemianopsia is caused by the focus extending into the visual radiations of that locality. There is no reason to assume that there is a continuation of this focus toward the occipital lobe, as one would have to believe according to the older theory—as to the localization of the lesion in these cases of hemianopic hallucinations. Neither is there any reason by which a deeper disturbance of the functions of the occipital lobe, besides the optic tracts, could be demonstrated. Thus, this first case can only be looked upon as one contrary to the older theory.

Regarding hallucinations in general, it is now known that their provocation, and I emphasize provocation in contradistinction to the question as to the seat of the causative processes, can take place at any part of the visual apparatus, from the cornea to the cortical layer of the occipital lobe. Undoubtedly a similar theory, upon a broader basis, regarding hemianopic hallucinations will eventually be promulgated.

CASE II.—Man, aged fifty-six years; always well; fell on his head from a sleigh about one year ago without any apparent harm. About four months before the first examination (February 16, 1902) he noticed that his left leg "went to sleep," also pains in left big toe; soon after a disturbance of vision toward the left was observed. Preceding the symptoms by two weeks he had occipital pains; other disturbances were not observed, especially no apoplexy. The patient afterward had a certain numbness in his left hand and for a few days after the disturbance of vision he noticed toward the left, in the blind half of the field of vision, all sorts of figures, or imagined occasionally somebody was sitting next to him. Dr. Bayer, oculist at Reichenberg, made the following examination under date of October 9, 1901:

"*Status Præsens.* Looking outward the right eye lags, pupils equally dilated, 3 mm. in diameter, illuminating right retina contracts slower than left (hemianopic pupillary reaction?), fundus normal. Binocular left-sided defect of field of vision with straight limitation, which only

[1] Liepmann and Kalmus. Ueber eine Augenmasstörung bei Hemianopikern. Berliner klin. Wochenschrift, 1900, No. 38.

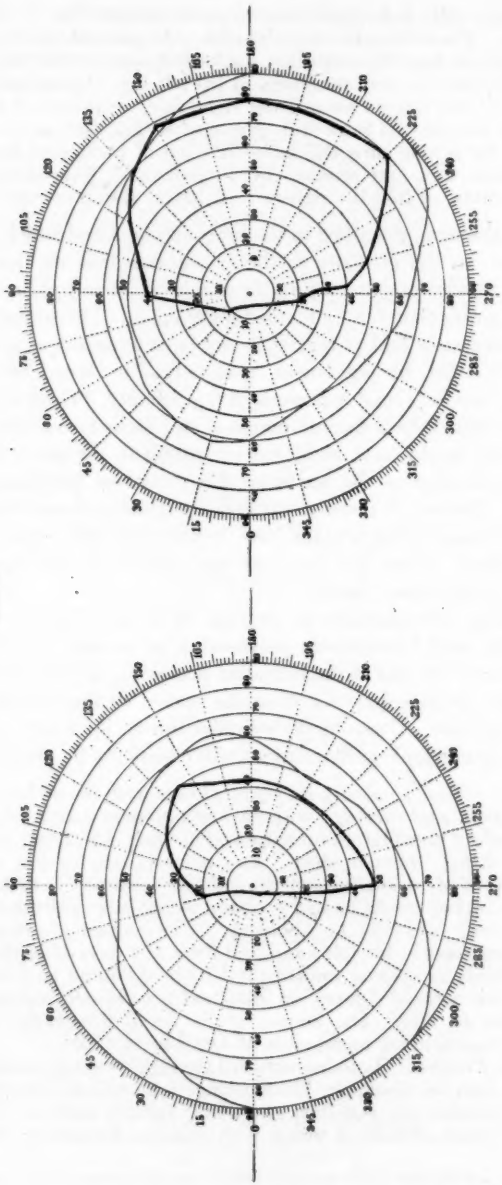

FIG. 2.

toward the middle deviates slightly toward the left. R. and L. 5/5 without correction, lead crust on his teeth, carotids feel somewhat harder than normal. Hemianæsthesia sinistra."

About one month later he had a seizure similar to an attack of Jacksonian epilepsy without loss of consciousness. Examination revealed, besides the hemianopsia, left-sided hemianæsthesia. My own examination on February 18, 1902, showed no disturbance of motion; the hand performs even the more delicate tests correctly. The sensibility of the hand seems slightly diminished, especially in fingers. Objects are not always definitely recognized. Sense of location undisturbed. Ophthalmological examination: Jaeger No. 1 can easily be read with glasses corresponding with his age. Insufficiency of right rectus internus; color sense normal. The right halves of both papillæ show changes in color Left homonymous hemianopsia with field of vision as seen in Fig. 2.

While the first case, when critically considered, admits of a possibility that the occipital lobe might be affected, in the second case this view must be altogether excluded, as moreover the provoking focus, producing the hemianopic hallucinations, is still further advanced and localized in the optic tracts. Looking at the symptoms of this case we will find only two localities by lesion of which the symptoms can be produced. We might consider the upper parietal lobe and the visual radiations running beneath it. But if we look upon the extent of involved territory, to make such extensive phenomena of hemianæsthesia and hemianopsia stationary, no apoplexy being present, we can only imagine that the latter were produced by one or several very small, very close foci of probable sòftening and we must look in another region. This we have in the posterior knee of the inner capsule, where the most posterior portion of the thalamus opticus meets the corpus geniculatum externum. In this region even a small focus can produce stationary hemianopsia or at first a complete and general, later incomplete and partial hemianæsthesia, as Henschen has demonstrated in a great number of cases.

Regarding the hemianopic hallucinations in this case, they present themselves as irritation phenomena, keeping step with the settling of the focus; while in the former case the identically localized hallucinations must be referred to some unknown irritative processes in the focus, the latter having existed for a longer period.

Case III. Woman, aged fifty-two years, has suffered for years from arteriosclerosis and cardiac disease, with frequent severe stenocardiac attacks with dyspnœa, pallor, and fainting. Had an apparently similar seizure, lasting for five minutes, on November 26, 1901, after which she suffered from a paresis of the left arm and left side of face. Disturbance on the left side of the field of vision was only noticed the second day and was then diagnosed by the attending physician as complete homonymous hemianopsia. Fundus normal. These attacks recurred several times, leaving a weakness of the former paretic muscles and occasional severe pains and paræsthesia in the left arm, left big toe, and

the left side of the face. In addition she volunteered the statement, that for a few days after the first attack she suffered from an indefinable delirious condition, characterized by hallucinations, showing in

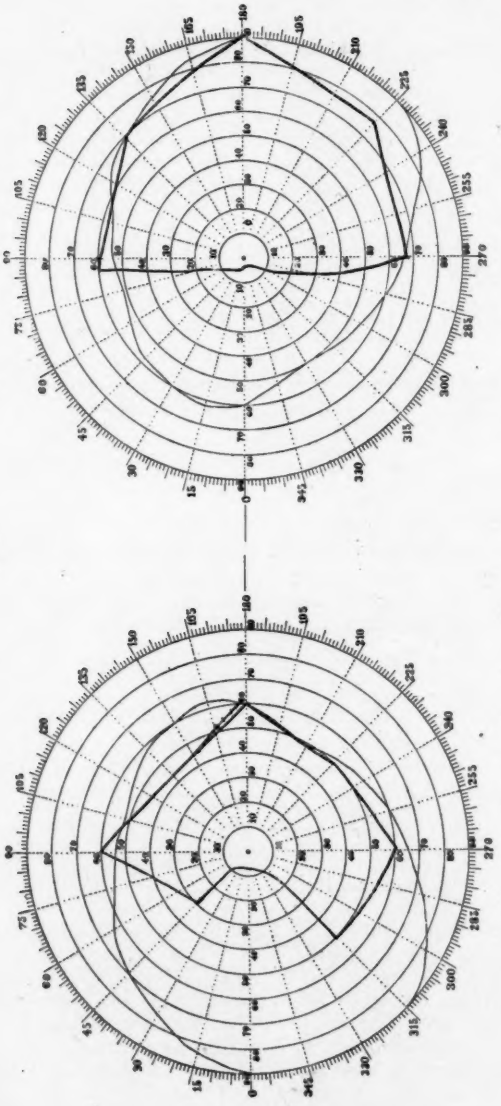

Fig. 3.

both the left blind halves of the field of vision all sorts of phantoms. She also asserted that at that time she had disturbances of smell and taste of a subjective nature, also sensations of coldness in the left half of the body. Urine was normal. Later had repeatedly pains in left arm, especially in the left index finger, and at the same time headache in the right side, dizziness, and feelings of anxiety. The left-sided homonymous hemianopsia remained stationary.

My examination on February 22, 1902, confirms the above-mentioned cardiac and arterial symptoms. Fundus normal. Complete left homonymous hemianopsia (Fig. 3). Sense of smell in left nostril diminished. Signs of paresis in the region of the left facial nerve, tongue not affected; slight evidences of paresis of the left hand, particularly of the thumb and index finger. Objectively no marked difference of motility of the legs, although subjectively complained of; sensibility in all qualities objectively quite normal; knee-jerk more pronounced in left than right. Subjectively, patient states that sense of touch and sensibility are diminished in the left hand.

The patient later complained of occasional headaches, accompanied by violent pains in the left index finger and left side of the neck, weakness of the left hand, which, however, disappeared after a few days. Later these attacks became less frequent. A subsequent examination showed the same picture of the field of vision. Liepman's disturbance still present.[1] Patient still complains of pain in the left upper extremity and a peculiar sense of dryness at the root of the tongue and at the palate.

Concerning this case I shall be very brief. The facts concerning localization agree so perfectly with Case II. that I should be forced to repeat. I believe, therefore, that according to all rules of localization the lesion should be considered as being in the most posterior part of the internal capsule, the optic thalamus, and perhaps also the corpus geniculatum externum. The hemianopic hallucinations here were also an expression of irritative processes produced by the focus, and later disappearing, when the irritation was removed. Hence this case also serves to strengthen the above cited theory of the localizing importance of hemianopic hallucinations.

I would call particular attention to two symptoms: 1. The evident "central" pains in the left arm. As early as 1879 Kahler and I[2] called attention to the relation of these pains to a focus in the region of the "Charcot carrefour sensitif." 2. Dryness of the mouth. Dejerine and Egger[3] have lately described a case of hemianæsthesia and concur with the investigations of Bechterew and Mislawsky, that the thalamus opticus exerts an influence upon salivation.

As an addition to the foregoing cases with stationary hemianopsia, I shall mention the observation of a case of scotoma scintillans bitem-

[1] I would here call attention to the first case. The man soon lost this disturbance and also understood how to correct his hemianopsia by holding the head properly, while this patient is as yet unable to do so.

[2] Beiträge zur Path. u. path Anatomie des Centralnervensyst., p. 42.

[3] Revue neurol., 1903, p. 402.

porale, justifying it for the reason that not only elementary light phenomena were present, but that the case progressed to real hallucinations. This case still further serves to strengthen the above theory.

CASE IV. Merchant, aged fifty-three years. Always healthy, except for "terrible" attacks of migraine, which lasted up to his thirtieth year and then disappeared. Strongly addicted to smoking, very moderate drinker. The somatic condition shows no abnormality of the nervous system, no evidences of alcoholism or pronounced atheroma. The eyes, frequently examined, are perfectly normal. Exposing himself to cold, he suffered from "rheumatism of the head," which nothing could alleviate. He felt "as if thousands of knives were in his head." As a last resort he wrapped his head in absorbent cotton, which made him feel very warm. In the evening of the same day, without any previous excitement, he noticed suddenly, without any pain and lasting for but a few seconds, on both external halves of the visual fields, peculiar phenomena, viz., colored lights, colored planes, figures, etc., while what really was in these halves of the fields he could not see. The line of demarcation between the lateral "blind" portions and the middle seeing ones was not sharp. As the patient happened to be in company, he did not pay any attention to these things, simply rubbed his eyes and everything vanished. On going to bed it suddenly returned so that on both temporal sides he noticed spots of soot which commenced to move; immediately after appeared numerous, rapidly moving objects of hallucinations, such as men, mice, serpents, birds. They were in all possible colors, blue, red, etc.

The very much frightened patient had sufficient presence of mind to observe the entire phantoms, and found just as before, that objects in the temporal fields of vision could not be seen and that, when trying to fix the phantoms, they immediately disappeared outward. Especially remarkable is the fact that he saw people of all shapes and sexes lying next to him.[1] He also observed that what he noticed in the temporal parts at times formed vertically running snake-like lines, so that, for instance, men in their upper parts moved in snake-like gyrations and in their lower parts formed into stationary curved lines. Closing the eyes did not change the phantoms. It then appeared to him as if he were looking into enormous mirrors. At one time he also observed that the real objects situated in the temporal fields of vision moved in the same snake-like lines. This all disappeared for a short time, but returned later and remained, without his being able to fall asleep, until the morning. He then undertook a journey to consult a specialist. But even then these strange visions did not disappear. While going to the station and throughout the journey he was accompanied by these hallucinations on the temporal sides of his fields of vision. "He lived in a panorama." The side cushion of the railroad car, on which he rested his head, changed into a human being, and later into a hundred other things. The figures appeared as a whole, but changed below into actively moving "fringes." Patient performed all his business obligations and consulted an oculist, who found the fundus normal and diagnosed "nerve trouble." Only toward evening, after having

[1] Compare Lauder Brunton (Journal of Mental Science, April, 1902, p. 253), Relations of Fairies and Other Visions to Scotoma Scintillans.

taken bromides, did the phenomena subside. Patient slept all night and the next morning the hallucinations still appeared, though less frequently and had lost their color, appearing black, and disappeared entirely during the day.

These observations during the attack of this very intelligent patient are so exact that nobody will fail to recognize the case as one of scotoma scintillans hemianopicum. But what makes the case in my opinion significant is the fact that we here have to deal with the bitemporal variety of that form, a variety which, as far as literature is concerned, I have not seen mentioned anywhere. Referring to the seat of the lesion provoking the process, I can only define one certain locality, viz., the chiasm. Although by reason of other observations there is no doubt in my mind that the phenomena of scotoma scintillans may be produced by exciting causes in the retina itself, there is no reason to assume that in this case an excitation in both temporal parts of the retina did take place; it is rather within the laws of scientific logic to assume that the phenomena were due to a lesion in one single locality. Lesion of the middle portions of the chiasm produce the now not quite so rare (*loc. cit.*) form of stationary bitemporal hemianopsia. Jolly has fully shown that the excitation provoking lesion for other forms of scotoma scintillans is to be looked for in the chiasm, and his view corresponds with this case. Whether we have to deal here with a hyperæmic condition of the hypophysis, the tumors of which especially produce acromegaly with stationary bitemporal hemianopsia,[1] it is hard to say. The possibility is also not excluded of having an abnormal vessel in the region of the circle of Willis. Weir Mitchell cites a case in which aneurism of an anomalous arterial branch produced stationary bitemporal hemianopsia.

A few more words in regard to the theory that we here have to only deal with a functional disturbance.

I believe there is no indication whatever of a localized basilar process, neither is there any reason to assume that the just mentioned causes, producing stationary bitemporal hemianopsia, can be applied to this case. We might still think of syphilitic basal meningitis, which has a predilection for just this locality and of which Oppenheim mentions bitemporal hemianopsia as an after-effect, but the etiology, the absence of any other endocranial disease, the sudden onset of the fully developed phenomena, above all absence of any ocular disease, all speak against it. There remains nothing, therefore, but the assumption of functional disturbance, which in this case is the migraine from which the patient suffered in former years. That he had no headache during or imme-

[1] Carven (Medical Chronicle, January, 1903) recently mentions a case of tumor of the hypophysis without acromegaly. This patient suffered from migraine a long time before and after the bitemporal hemianopsia had been demonstrated.

diately before the optical phenomena can not be considered here, as we know that frequently a typical attack of scotoma scintillans is unaccompanied by headache. Moreover, it is not clear in what relation the "rheumatism of the head" stands to this case. There is also absolutely no reason to believe that the phenomena were the premonitory symptoms of a subsequent paralysis or tabes.

In conclusion I would point out the transition of the elementary light phenomena of scotoma scintillans into real hallucinations, again confirming the afore-mentioned theory, that hemianopic hallucinations may be produced by a variety of localized excitations in the optical tracts and not in the occipital lobe only.

A CASE OF SPONTANEOUS PROLAPSE OF BOTH LACRYMAL GLANDS. REVIEW OF THE LITERATURE.

By Dunbar Roy, A.B., M.D.,
CLINICAL PROFESSOR OF EYE, EAR, NOSE, AND THROAT DISEASES IN THE ATLANTA COLLEGE OF PHYSICIANS AND SURGEONS, ATLANTA, GA.

The case here reported is anomalous in character rather than of any practical interest. In looking up the number of similar cases which had previously been reported, and also any other literature bearing upon the subject, I was surprised to find that spontaneous prolapse of the lacrymal gland could be placed in the category of rare affections.

Several cases of traumatic prolapse have been reported, as also one or two cases of spontaneous prolapse of one lacrymal gland, but nowhere can I find the report of a case where both lacrymal glands have become spontaneously detached.

Mary M., colored, aged twenty-seven years and unmarried, presented herself at the clinic of the College of Physicians and Surgeons in September, 1902. She came with the complaint that both upper eyelids remained swollen since an attack of bronchitis three weeks previously. There was no pain and no interference with vision; practically no discomfort from the present condition.

On casual inspection the upper lids gave all the appearance of œdema, especially on the temporal sides. By palpation I found a well-defined glandular body just beneath the border and at the outer edge of the supraorbital ridge. It was especially prominent when the patient looked down and held the head forward. Both sides had absolutely the same objective appearance and symptoms. It was freely movable under the touch, and could be pushed up underneath the orbital rim. The patient was perfectly positive that there was nothing wrong with the eyes up to three weeks previously. No enlarged glands could be found in any other portion of the body. As the condition was giving the patient no trouble, she refused any operative interference, leaving out the question of its own advisability. When last seen there were no changes visible.

I was very confident, as were my colleagues, that the bodies felt were the lacrymal glands, but we could in no wise account for this dislocation. There was no history of traumatism to which it could be traced, nor any severe straining on the part of the patient except the previous cough.

In this latter respect, as well as in several others, the case is similar to the one reported by Snell in 1881.

Anatomists tell us that the lacrymal gland is enveloped in a capsule which, by its attachment to the periosteum of the orbital cavity, is retained in place. In a case of this kind we can but surmise some anatomical malformation or some relaxation of the surrounding tissues as a cause of such a pathological condition.

In looking up the literature of this subject but few cases could be found recorded. For this reason I will give in detail a résumé of all cases of both spontaneous and traumatic prolapse of the lacrymal gland of which I have been able to find any report.

CASE I.—In the *Ophthalmic Review* for the year 1881–82 we find a case reported by Simeon Snell, as follows:

On March 7, 1882, I was consulted by a tradesman, aged forty-five years, in consequence of a lump in the left eyelid. It had been observed first a week previous, and had been seen by Dr. Brook, who sent him to me. The swelling was distinctly visible, and particularly noticeable to the touch. Situated in the upper eyelid at its external part and coming from under the frontal bone, it was felt beneath the structures of the lid as about the size of an almond. It could be pressed between the fingers, but readily slipped back into the orbit. Its surface felt more smooth than irregular. Pressure causes it immediately to recede into the orbit beneath the frontal bone, but after depressing the head it was again found to be visible in the lid. This he had himself observed, and after several attempts to replace it he always found on bending the head downward that it had reappeared. He complained of it causing him discomfort, which was increased on manipulation.

On the night of the first appearance of the substance in the eyelid he had gone to bed in no wise ailing, and had detected it on rising in the morning. He had in the night been coughing a great deal. The same side of the head is marked by a large venous subcutaneous nævus which extends to the eyebrow and apparently passes into the orbit. The man is a free drinker, and is frequently laid up in consequence.

The question as to what the "lump" was did not present any great difficulty. Its situation, size, and feel suggested directly its being the lacrymal gland displaced, and this was the diagnosis also of the medical man who first examined the patient.

It was decided to try the effect of compression in keeping the gland in place. A few days later when the patient presented himself he desired to postpone the wearing of a "pad" for a short time. I, however, pressed the gland well back with my finger and kept it replaced. He left me without its having reappeared.

Being soon after laid up with one of his attacks of illness, he did not again see me until five or six weeks later. The swelling in the lid

was then no longer visible, and he asserted that it had not appeared since his last visit, and that the sense of discomfort had gone.

Spontaneous displacement of the lacrymal gland must, I imagine, be a very rare condition. I cannot recall a recorded case like the one described. I remember the case of a man under treatment for keratitis some years since in whom the glands were distinctly visible and freely movable in the eyelids. The condition was congenital.

I am further reminded by a gentleman attending my clinic of another similar case which came under observation since the one just referred to.

It is worthy of mention that in the case of my patient, in his attacks of illness following his drinking bouts, he sometimes suffered from epileptiform convulsions, during which the large venous nævus at the margin of the orbit became greatly distended, looking as though it were ready to burst.

Case II. Reported by Wm. George Syme from the hospital practice of Dr. Argyll-Robertson in the *Edinburgh Medical Journal*, 1887, vol. xxxiii.—J. C., a man aged thirty-six years, applied for treatment in May 1887, suffering from ptosis and a tumor situated in the right upper eyelid. The tumor extended along the whole length of the upper lid, from a point above the inner to a point above the outer canthus. By its weight it caused ptosis; the eyelids were nearly closed, and the upper lid could hardly be raised at all, the mass catching at once on the orbital edge. The skin over the tumor, though slightly congested, was not œdematous, and was freely movable over it. The tumor, which felt firm, lobulated, and glandular, was not attached to the deeper structures except about the middle for a distance of one-third of an inch, where it was moored to the orbital edge of the frontal bone.

It could not be reduced into the orbit. The ocular movements were in no way affected, and the vision in the two eyes was the same. The tumor was perfectly painless even on firm pressure; the neighboring lymphatic glands were not enlarged, and the patient seemed to be in perfect health. The history of this affection was that about the end of January of this year the patient had first noticed the swelling about the size of a bean in the upper eyelid. This was painless, but troublesome on account of the ptosis which it caused. At this time also he noticed that the right eye was more watery than the left—a condition which had not previously been present, and the swellings were larger in the mornings and became rather smaller in the day. He thinks that the tumor had suddenly attained the size of a bean, but is not quite certain; at any rate it had steadily increased in size from the time of its first appearance. In the winter of 1885 the patient had noticed a similar swelling in the same situation; this however had been much smaller, and caused no annoyance, and after a few weeks spontaneously disappeared.

Under these circumstances the diagnosis was made of a displaced and chronically enlarged lacrymal gland. On May 10 an incision was made over the tumor in the long axis of the upper lid, and the

gland, which was lying immediately under the skin, was turned out. The accessory portion of the gland was found lying in the proper situation of the main part, and was left to carry on the usual functions of the gland. As soon as the swelling had subsided there was a marked diminution in the ptosis, which continued to improve until the patient left the hospital.

CASE III. Reported by Dr. Henry D. Noyes and published in the *Transactions of the American Ophthalmological Society*, 1886–87.—Miss Carrie S., aged twenty years, of Paterson, N. J., presented herself February 8, 1887, because of the swelling under the upper lid of the right eye which had been growing for nine years. For the last few months it had been uncomfortable. It rested upon the eyeball at its superoexternal side, and reached within 6 mm. of the cornea. It was flattened, not lobulated, and nearly circular in outlines, and merged gradually into the surrounding conjunctiva. A few vessels coursed over it, and it had a faint yellow color. It was nearly three-fourths of an inch in diameter. It could be moved freely upon and also moved with the globe. It was very compressible and elastic. It seemed to have a capsule which at its lower border was densely white and firm; over the remaining surface it was thin and translucent, but with a yellow tinge. There had never been any blow nor sign of disease in this region, nor inflammation, nor serious pain. The progress had been very gradual and the cause unknown. The other eye had been lost sixteen years before by accident. This globe was atrophied, yet on lifting the upper lid a rounded mass, red and slightly lobulated, and a little larger than a cherry-stone, appeared at the upper and outer angle of the orbit, and was regarded as a part of the lacrymal gland which had become displaced.

Treatment. An incision was made obliquely across the tumor and the conjunctiva dissected off by the closed blades of curved and sharp-pointed scissors. Very little cutting had to be done, and the hemorrhage was moderate. When the mass was separated it proved to be normal lacrymal gland.

The conjunctival wound was stitched and a bandage applied. The healing was prompt and the reaction moderate. A wrinkled and swollen condition of the conjunctiva remained for a few weeks at the site of the operation. The prolapse seems to have been caused by simple laxity of the enclosing fibrous capsules, without any propulsion from behind.

CASE IV. Reported by L. Mauthner in *Wien. med. Presse*, 1878, p. 110.—Man, aged twenty-four years, came to the clinic October 4, 1868, for inflammation of the eye of four weeks' standing, and for a tumor in the upper eyelid, first noticed a few days before. There was an injection of the conjunctiva palpebrarum et bulbi, ciliary injection, and diffuse coloring of the cornea; the upper lid was much thickened and drooping, and one feels in its lateral half a hard, movable, and slightly rough, painless tumor, not adherent to the skin, of size and form of the lacrymal gland. This showed no sign of an orbital tumor. By the use of atropine and compress bandage the inflammatory phenomena of the cornea disappeared in five weeks, but the tumor of the upper lid, which Jaeger thought might be the lacrymal gland, had not changed in spite of using iodide of potash and iodine salve. Jaeger operated November 11 1868; laid bare the tumor by section,

from in and above outward and somewhat down in the direction of the length of the tumor; a small part removed showed nothing abnormal. The gland was replaced in the orbit and made fast by sutures, the patient being discharged sixteen days after operation. The gland was fixed to surrounding tissues and only a small tumor could be felt at the lateral upper orbital edge.

CASE V. Reported by S. S. Golovine, *Clin. Maitop.*, Mosk., 1895, vol. v. pp. 930–941.—A young man, aged eighteen years, admitted to the hospital July, 1893. The upper eyelids seemed swollen and slightly drooping. He carried his head erect, and even from time to time threw it backward, as if he wished thus to compensate for the fall of the eyelids; he instinctively contracted the frontal muscle; from this cause the skin above the brows was constantly wrinkled, and the face had a very strange expression. The skin of the eyelids was tense, very delicate, and formed a fold almost transversal which came down on the eye. This was not equally pronounced in its entire extent. The crease which one generally finds below the orbital edge was effaced. The skin, of normal color, was transparent and allowed subcutaneous veins to be distinguished. These phenomena were symmetrical, but more pronounced on the left eyelid. Below the skin one might palpate a hard compact lump, like a grape, ovular, 1½ cm. in diameter. It could be displaced in all directions and slid easily across the anterior surface of the cartilage. These movements seemed limited toward the interior by some adherences, although it was impossible to feel them. The movements were freer upward and outward. When the finger was raised the body came back little by little from the orbit, falling into the part of the cutaneous sac, which covered the external commissure. The elevation of the lid was hindered and the patient overcame this difficulty by contracting the frontal bones. It was very easy to turn the lid back, the mucous membrane of which was altogether normal.

The lacrymal conduits were normal, although latterly the tears were less abundant. Although the other organs were not changed, the general organism seemed much depressed and its development much retarded. The lids had been changing for the last three years.

There were two methods of treatment: 1. Total extirpation of the gland. 2. To bring the gland back to its place and fix it. Both methods were used.

The writer began by removing the right lacrymal gland.

The result of the operation was very satisfactory, the wound healing by first intention. Eight days later the other eye was operated upon, the lacrymal gland being replaced in the orbit and fixed with sutures. The result was very good, and three days later the cutaneous sutures were removed, and six days later those which held the gland in position. Two years later the writer received a letter from the patient, who expressed himself as well pleased with the result.

CASE VI. Reported by Briere in *Bull. et mém. de soc. de chir. de Paris*, 1876, vol. ii. p. 592.—Boy, aged eleven years. The affection began five and a half years ago. He struck his left temple against a nail. The wound suppurated and the external wall of the orbit began to necrose. The skin of the eyelid, temple, and brow, being drawn back by the cicatrix, was depressed to the walls of the orbit, which had become angular and unequal. The free edge of the eyelid, drawn more

and more up and out, took progressively a form characterized by an elongation of the free edge, which measured 22 mm. more than that of the healthy lid. This deformity, produced by numerous cicatricial bands, had, as a secondary result, compressed the lacrymal gland. This gland was not protected by the bony depression in which it was normally lodged. The opening of the lid, increasing more and more, the lacrymal gland was soon exposed. The lobules, not being compressed from this period, became larger and formed a tumor the size of a large hazelnut, between the lid and the globe.

Operation August 10th under chloroform.

1. The writer took the exact length of the healthy right lid, and from the internal angle of the diseased eye he measured this length on the elongated lid.

2. Incision between lid and lacrymal gland to mobilize the upper lid.

3. Dissection of the upper lid 4 to 5 cm. as far as the middle of the forehead; considerable bleeding.

4. Curved incision, separating the outer side of the lacrymal gland from the superfluous part of the lid which was to be excised.

5. Incision at 2 mm. outside of the ciliary line and parallel to the preceding except at the upper part.

6. Excision of the whole flap.

7. Dissection of the temporal flap, the longest and most difficult part of the operation.

8. He assured himself that union would be possible by sliding the two flaps.

9. Before applying the sutures he put on a compress bandage and allowed the patient to revive from narcosis.

10. Twenty minutes later, when hemorrhage had stopped, the lacrymal gland was replaced in the cavity which had been made by the removal of the sequestra of bone.

11. Applied twisted sutures with fine pins.

The operation may be considered a mixture of that of Adams and Jaeger.

It is now eight months since the operation. The lid covers the eyeball except the parts nearest the external commissure, where there is a space of 2 mm. between the lids when the lids are closed. The lacrymal fistula has closed spontaneously; there only remains a slight depression of 2 mm. in length, the bottom of which is healthy looking.

Case VII. Reported by G. Ahlstrom, in *Centralbl. f. prakt. Augenhlk.*, 1898, vol. xxii. p. 300.— The patient, a boy aged twelve years, fell when he was three years old and struck his right eye upon a sharp-cornered block of ice. There was a bloody wound which soon healed. When first seen there was found to be quite a high-grade ptosis of the right eye, so that in looking straight out the upper lid covered half of the pupil. The ptosis had been caused by the thickness of the lid. The skin of the lid was normal, easily movable, and hung on the palpebral edge in the form of a fold a little below the edge; no scar was to be seen. By palpation one could perceive a tumor-like mass just below the skin, about the size of an almond. This was quite firm, somewhat ragged, showing no adherence with the deeper parts, but was easily movable under the skin, and could not be pressed into the orbit. The tumor was removed. It was found to be the lacrymal gland.

The ptosis became less immediately after the operation and the lid resumed its normal appearance.

CASE VIII. *Bericht d. k. k. Krankenh. in Wien*, 1889.—A very superficial mention is made of this case. A laboring man, aged forty-one years, was transferred to the ward. He had suffered from a scrofulous (?) conjunctivitis, and also had had an ulcer of the same nature on the upper eyelid. As a result of this latter there was a depression and strangulation of the lacrymal gland and ectropion, by which the upper lid adhered in about its middle to the edge of the orbital cavity as a part of the lacrymal gland, being in this way removed. The remainder of the gland is seen as a strangulated tumor. Separation of the adherence to the edge of the orbit, the transplantation of the flap from the temple region, resulted in a cure.

CASE IX. Reported by von Graefe, *Arch. f. Ophthalmol.*, 1866, vol. xii. p. 224.—Boy, aged ten years, was brought to Graefe on account of a wound of the upper eyelid, having been struck the day before by falling on a piece of glass. There was a wound 18 mm. long which passed through the whole thickness of the upper lid parallel with the edge of the orbit. From the wound there arose a reddish plug about the size and shape of a finger phalanx, which he recognized as the lacrymal gland. It hung down over the cleft of the lids, which it partly covered and was somewhat drawn in at its base, so that the physicians thought of removing the whole mass. The prolapsed lacrymal gland was held back by an assistant while Graefe closed the wound with four stitches and then applied a compressive bandage. There was a slight suppuration, and in the end there was left a fistula of the lacrymal gland.

CASE X. Mentioned by Panas in his *Lecons sur les Affections de l'appareil lacrymal*, Paris, 1877, p. 8.—A case similar to that of von Graefe was shown by Lariboisière at a clinical meeting. There was a deep contused wound in the left upper lid just at the external extremity of the orbital ridge, in which was seen a prolapsed lacrymal gland. A single stitch of metal suture was enough to keep the gland in place and bring on cicatrization of the edges of the wound, which was 2½ cm. long. When the patient left the hospital there was a slight induration of the gland which would probably disappear in time.

CASE XI. Reported by R. Rampoldi, *Ann. di Ottamol.*, Pavia, 1884, vol. xiii. pp. 68–70.—Boy, aged twelve years, admitted to the hospital in January, 1880, for grave ectropion of the upper lid. About a year before he had been struck by a stick in the left temporoparietal region, followed by pronounced exophthalmos. He was taken to an eye clinic, where a diagnosis of retrobulbar phlegmon was made, the abscess opened, and the exophthalmos thus much reduced. The vision was preserved intact, and the patient returned home almost completely cured. A slow process of the osteoperiostitis went on in the orbit, the drainage aperture becoming fistulous and discharging slowly disintegrated grayish pus, while the cicatrix adhering to the brow of the orbit turned back the aperture of the fistula and drew the lid strongly upward and outward, turning the free margin so that the externo-superior conjunctiva was all turned out. The patient, in a very anæmic state, was now admitted in the hospital. The exophthalmos was moderate and the bulb turned out in a limited degree only. The cicatrix was strongly adherent and had a tendency to invade the lac-

rymal fossa. A local treatment was used with disinfectants and the Paquelin cautery, supplemented with a liberal diet and tonics. After some time the patient left the hospital before it was thought best to attempt a radical cure.

In August, 1880, the palpebral ectropion was considerably increased, and at the site of the wound was seen a tumor of solid consistence of the size and shape of a wild mulberry, which was recognized as a dislocated lacrymal gland. As it was impossible to replace it, the tumor was destroyed with the needles of Boerelli.

CASE XII. Reported by R. Hilbert. *Klin. Monatsb. f. Augenheilk.*, vol. xxxviii. p. 478.—Boy, aged one and one-half years, was brought to the writer December 28, 1889, whose left eye was said to have been injured the day before by his falling on a beam. Close to the outer left lid commissure just under the edge of the orbit was a wound 1 cm. long, horizontal, and from which protruded a red roundish body about the size of an almond. The whole lid was black and blue. From the article of Ahlstrom the writer considered the tumor to be a lacrymal gland. He removed it with one stroke of the scissors, closed the wound with two stitches. Healing followed promptly; there was no ptosis, and the microscopic examination showed the correctness of the diagnosis.

CASE XIII. Reported by Goldzieher, *Jahresber. u. b. Leist. u. Fortschr. im Gebiete der Ophthal.*, Tübingen, 1878, vol. vii. p.466.—A child, aged one year, was wounded in the upper eyelid, which was followed by bleeding and the appearance in the wound of a dark-red, fleshy body. When the writer first saw the patient this body was the size of a hazelnut and completely filled the skin wound, which was 4 mm. long. This body was cut off with the scissors and the wound healed promptly. An examination of the growth removed showed it to be the lacrymal gland.

CASE XIV. Reported by Haltenhoff, *Annales d'oculistique*, 1895, pp. 319–321.—This was the case of a boy, aged two and one-half years, who fell forward in a road filled with broken stones. When he got up he could not open his eye, and in the lid appeared a wound from which protruded a solid bleeding mass. The upper lid was bandaged, and three days later was seen by the writer. The upper lid was found to be falling over, œdematous, tense, and quite ecchymotic. In its external third, several millimetres above the free border, there was a sort of fleshy protuberance of a livid grayish-red color and of a consistency which seemed to be firm for a simple clot. It adhered by a sort of pedicle to the bottom of a horizontal wound of the skin. The patient was etherized and the palpebral fissure opened enough to prove the integrity of the eyeball. It was then decided that it was a hernia of the lacrymal gland. The sharp edge of a broken stone had cut the lid wide open, penetrated obliquely from below upward through the wound. The three days' strangulation rendered it difficult to reinstate the protruded gland, so the author profited by the narcosis and cut it off from the lid. It was then noticed that at the bottom of the wound there were several grayish-red lumps, apparently lobules of the gland. They belonged to the palpebral portion which is formed by a mass of little isolated lobules with loose connections. The wound was united with two silk sutures, dressed with dermatol, the wound closing by first intention except at one small point. The recovery was perfect,

and no difference was noticed in the appearance and moistening of the two eyes. Microscopic examination by Dr. Buscarlet confirmed the diagnosis.

CASE XV. Reported by L. Pistis in *Annales d'oculistique*, 1895, pp. 468–470.—J. D., aged one year. The child had fallen on her face in a road filled with stones, and the lid was cut near the external orbital border. The wound was on the upper lid of the left eye, and was œdematous and ecchymotic. The skin was intact only near the internal portion; the remainder was a bleeding surface with an almost triangular flap detached from the external third of the orbital border and turned downward. Through the lips of the wound, by a short pedicle, there appeared an oval gray mass. On raising the lid the writer found the cornea intact, and there was no exophthalmos. The gray mass was the orbital lacrymal gland. The writer reduced the prolapsed gland by suturing the lid in front of it, his observation differing in this point from that of Dr. Haltenhoff, who excised the protruding gland. Union took place by first intention, and as a result the child has only a slight prominence in this region as if the gland had not perfectly resumed its anatomical position.

CASE XVI. Reported by W. F. Mittendorf, in *Transactions of the American Ophthalmological Society*, 1901, p. 382.—A young lady, aged twenty years, in pretending to be hypnotized by her brother, fell forward and struck his hand in such a manner that his index finger struck the upper part of the outer corner of her left eye. The next day the young lady found that there was a swelling in the upper temporal part of the eyeball, which hardly showed with the lids in the normal condition, but became very apparent by raising the upper lid. As the swelling did not diminish, the patient consulted the writer. The eye showed apparently no injury except a slight scratch of the finger-nail near the inner corner, the rest being perfectly normal, and so was the vision of the eye, nor was the motility interfered with in any way. The tumor itself was slightly painful to the touch and could be pushed up a little, but descended immediately as the pressure exerted upon it was relaxed. The upper and the outer part of the orbit felt a little tender, but no perceptible difference of the two sides could be made out. The diagnosis of dislocation of the lacrymal gland was immediately suggested from these symptoms.

The treatment of the case was simply expectant, as pressure failed to replace the swollen part and as it caused no visible deformity. The swelling has remained practically the same now after a lapse of three months.

The diagnosis of these cases of dislocated lacrymal glands is apparently easy, and especially so when there is any incised wound permitting easy inspection in addition to palpation.

In all of the cases reported, except the one by Mittendorf and Noyes, the deformity was noted externally rather than internally beneath the upper lid. In my own case nothing abnormal could be seen by the closest inspection beneath the lid.

Cases of spontaneous dislocation of the gland will, of course, present but one or two symptoms which are found when the conditions are due

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to trauma. With the exception of the deformity due to swelling and a possible limitation in the raising of the lid, these spontaneous cases usually cause no inconvenience to the patient.

It is a noteworthy fact that in none of the cases recorded has the lacrymal function of the gland been impaired, for in my own case neither an increase nor a decrease of the moisture could be noticed. Another remarkable symptom mentioned only in the case reported by Haltenhoff, where even the greater portion of the gland was removed, was the fact that there was no difference in the moisture in the two eyes.

The question of treatment must of course vary according to the character of the case. In those dependent on traumatism, where there is a contused or incised wound complicating the condition, operative intervention must always be considered. In those cases where there is no wound, or in the so-called spontaneous dislocation, the question of operation must be decided by the character of the individual case.

Mechanical compression is difficult of application. If the deformity is not marked and there are no uncomfortable symptoms, I do not see why it is necessary to do anything whatever. If there is ptosis from the presence of the gland, or if the latter should be painful and of great discomfort to the patient, fixation should first be attempted, and if that failed, there is no reason why the gland should not be removed.

ACUTE SUPPURATIVE THYROIDITIS.

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Acute suppurative thyroiditis is a very rare condition, and usually secondary to some infectious disease. As a primary condition it has only been recorded in a very small number of cases. A case belonging to this class came under the writer's observation and gave the following history:

F., aged forty years, a native of Italy, laborer by occupation, was admitted to the service of Dr. Parker Syms, at the Lebanon Hospital, on November 26, 1902. On account of the patient's inability to speak English, a good history could not be obtained. As far as could be learned, the patient was suddenly taken with a chill and high fever on November 23, 1902. He became gradually worse, and after two days was brought to the hospital. On admission his temperature was 103.2° F., pulse 128. He seemed in a state of profound sepsis, sweating profusely and being decidedly cyanosed. On examination, a diffuse swelling was found around the anterior portion of his neck, extending from the suprasternal notch to the upper border of the cricoid cartilage. There was some redness around the most prominent portion of this

swelling, but no fluctuation. The mass was tender, and engorged venous channels could be seen around the shoulders. The patient was breathing badly and swallowing with difficulty. There seemed to be considerable difficulty, also, in moving the head. No other lesion could be found, and a diagnosis was made of acute thyroiditis of unknown origin. The patient's condition being very grave, an immediate operation was decided upon, and under chloroform anæsthesia an incision was made by the writer, extending in the median line from the thyroid cartilage to two and one-half inches below. The muscles were separated, and the thyroid, readily exposed, presented itself as a smooth, dark-blue body, with a dull capsule. On opening the left side a few drops of pus escaped, and on thorough exposure the gland showed areas of necrosis. Iodoform gauze drainage was established, and the operation quickly terminated on account of the desperate condition of the patient. The temperature rose higher and reached 107° F. In spite of active stimulation, the condition became worse, and the patient died on November 27, 1902. The writer is indebted to Dr. Edward Schnapper, pathologist to the hospital, for the following record of the autopsy:

November 30, 1902. Body well nourished; rigor mortis well marked; no external marks of violence. Incised wound, two and one-half inches long, in median line of neck and leading down to thyroid gland.

Pleural Cavities: Normal, no fluid or adhesions.

Lungs: Right lung, upper lobe congested, middle and lower lobe congested and œdematous; left lung, congested and œdematous.

Heart: Left ventricle, wall thickened, myocardium pale in color, soft and flabby; cavity small. Right ventricle, wall thin, cavity slightly increased in size; mitral valve thickened and has small fibrinous nodules upon its edges. Aortic valves show moderate atheromatous changes. Pulmonary and tricuspid valves normal. Aorta shows here and there slight atheroma.

Abdomen not distended, and contains no fluid.

Omentum not adherent and contains a moderate amount of fat.

Intestines normal.

Appendix normal.

Liver moderately enlarged, its outer surface appears opaque, grayish-yellow, lobules indistinct.

Spleen enlarged, soft, Malpighian bodies prominent.

Right kidney enlarged, soft, capsule not adherent; cortex pale, swollen, pyramids congested. Both kidneys alike.

Bladder distended and normal.

Teeth and Gums: No pathological condition found.

Tonsils normal.

Tongue normal.

Œsophagus normal.

Stomach shows some post-mortem changes, otherwise nothing abnormal.

Pharynx normal.

Thyroid Gland: Both lobes enlarged and swollen; the right lobe is enlarged to a greater degree, firm, but in places there are areas of softening. In the right lobe there is an incised wound leading into a small irregular cavity.

Microscopic Examination of the Thyroid Gland. The connective tissue was found infiltrated with large numbers of leucocytes, the

polynuclear predominating. By this marked infiltration of cells the colloid containing acini are obliterated. In places there are areas of necrosis and abscesses. Inoculations upon agar from the thyroid gland showed a growth of pure streptococci.

From a study of the history and post-mortem record we are led to consider this case as one of primary suppurative thyroiditis. Inflammation of the enlarged thyroid is not at all uncommon, and cases of strumitis are seen comparatively often. Simple thyroiditis is occasionally observed, and the writer recalls seeing two cases belonging to this class; both of them occurred in women, were associated with constitutional symptoms, and terminated in resolution.

Suppurative inflammation of the thyroid, on the other hand, is a rare disease, and when it does occur it is usually secondary to one of the infectious diseases, or it may be due to an injury and consequent infection. Inasmuch as the thyroid is not frequently the seat of injury, the cases belonging to this class are of passing interest only. Ransohoff[1] calls attention to the fact that the thyroid has no excretory duct, being surrounded by a firm capsule, and is well protected against infection. It is most likely invaded by microbes through the blood circulation. According to Mygind,[2] quoted by Robertson in a paper on "Thyroiditis Complicating Typhoid Fever,"[3] the condition is most common in females between the second and third decades, rarely in previously healthy persons, or as a result of traumatisms, but most frequently during or following an attack of typhoid fever, acute articular rheumatism, diphtheria, influenza, malaria, erysipelas, puerperal fever, sepsis, orchitis or prostatitis. It may occur during or following scarlet fever, pneumonia, and pyæmia. According to Robertson, thyroiditis is most common after rheumatic or typhoid fever, and the first case supervening on rheumatism was reported by Molière, of Lyons. As a complication or sequel to typhoid, it usually terminates in suppuration, and this may be caused entirely by the Eberth bacillus. Thyroiditis may terminate in resolution, suppuration or necrosis. The simple form usually terminates in recovery; where gangrene occurs the condition is more grave, the symptoms being very marked, and this is usually due to a streptococcus infection.

In the subject of this report the infection was due to streptococcus, but the source of the infection remains undetermined after a careful autopsy which included a very careful examination of the mouth and pharynx. Rheumatism may be excluded because none of the joints showed any evidences of it during life, nor did the patient complain of

[1] Transactions of the American Surgical Association, 1894, p. 275.
[2] Journal of Laryngology, Rhinology, and Otology, 1895, vol. ix. p. 181.
[3] The American Journal of the Medical Sciences, 1902, vol. cxxiii. p. 67.

any pain. It seems worth noting that the right lobe of the thyroid was involved more than the left. It would be difficult to draw a clinical picture from the study of a single case, and the writer will not attempt to do more than point out that in his case the symptoms of sepsis were pre-eminent. The local signs might have been those of an ordinary phlegmon, but the constitutional symptoms were too severe. These might possibly be attributed to interference with the thyroid secretion. Dyspnœa, dysphagia, the movement of the swelling with the trachea, are usually present at some stage of the disease. In primary suppurative thyroiditis, early incision and drainage is imperative. That it was of little benefit in the writer's case can be accounted for by the very profound state of sepsis at a comparatively advanced stage of the disease.

A CASE OF COMPLETE BILATERAL DUPLICATION OF THE URETERS.

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(From the Anatomical Department, University of Texas.)

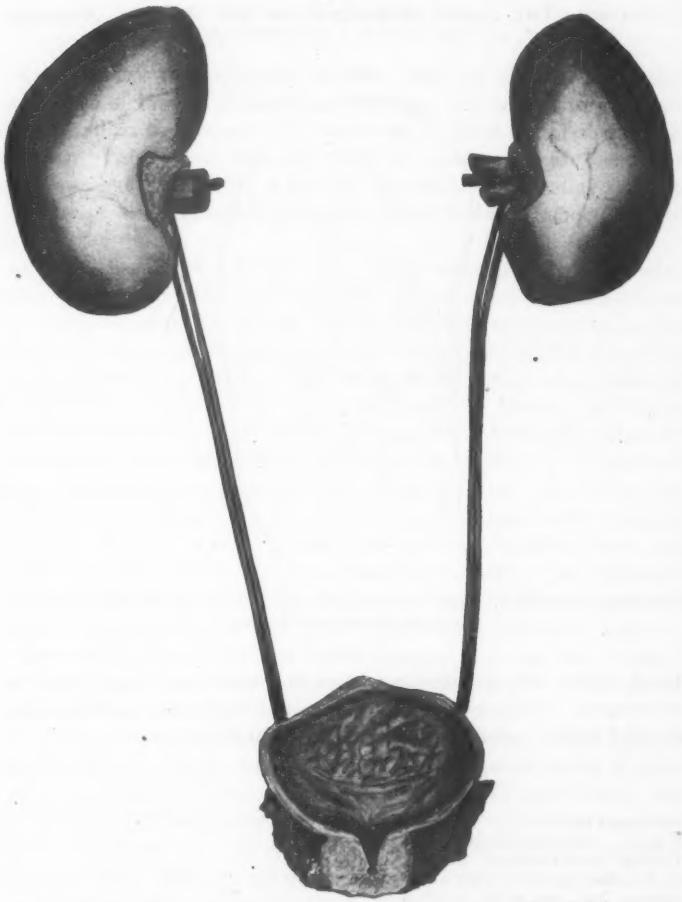
The following case of double ureters was first noted in November, 1902, but many things prevented its being recorded at that time.

The subject was W. T., male, negro, who died at Sealy Hospital of amœbic dysentery. Strongyloides were also found in his stools. Acute nephritis was present; but in this regard the double ureters, of course, had no clinical significance. However, had catheterization of the ureters or operative interference upon them been demanded, the double condition might have caused considerable embarrassment.

The kidneys measure 13 x 6½ x 4 cm. (right) and 11 x 6 x 4½ cm. (left). The left ureters are 25 cm. in length, and the right are 24 cm. All four are about the same size, and each is almost as large as an ordinary single ureter. Their relations to the vessels at the kidney hilum and to structures elsewhere in their course are normal. The bladder orifices, two on each side, are 1 cm. apart; the mesial (adjacent) ones being 2½ cm. apart. Dissection of the pelves and calices show that the upper ureter drains the upper third of the kidney, while the lower one drains the lower two-thirds. Fig. 2 is a tracing from a skiagraph of another kidney. It shows a bifid pelvis and the part of the kidney drained by each half; this resembles rather closely the condition found in each of the kidneys under consideration. The difference was that there were two pelves in each kidney, and one ureter from each pelvis. Moreover, the upper ureter drained rather less of the kidney than the upper segment of the bifid pelvis in Fig. 2. The two upper ureters open into the bladder by the two mesial orifices, the two lower ones by the two external.

It would have been interesting to have injected the infundibula and calices with bismuth and have taken skiagraphs, but the subject was

FIG. 1.



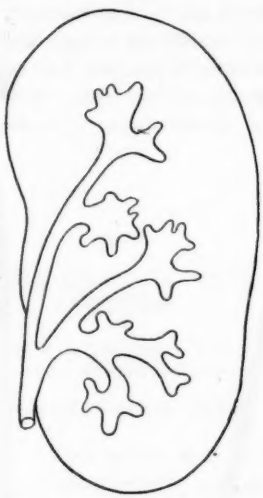
Shows kidneys, ureters, and bladder, some fat about the hilum on each side; one vein and one artery on the right, and two veins and two arteries on the left. The four bladder orifices are seen lying on a line slightly curved, with its convexity toward the urethral orifice. Surrounding the neck of the bladder is the prostate gland; and on each side of it is seen a mass of fatty tissue. (Drawn to scale from specimen.)

used in the dissecting-room, where the kidneys were too mutilated to admit of such measure.

Since it is the purpose of this paper merely to report this case, discussion of the embryological fault responsible for the abnormality is not entered into.

The rarity of complete bilateral duplication is shown by A. H. Gould, of Harvard (THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES,

FIG. 2.



Tracing from skiagraph of another kidney injected with bismuth, showing bifid type pelvis and usual arrangement of calices.

March, 1903). He reported two cases, and found only eight others in the literature. Unilateral duplication, partial duplication on both sides, and bifid kidney pelvis are not uncommon findings.

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In addition to these, I find a reference to two cases of "Complete Double Ureter" reported by Adami and Day (Montreal Medical Journal, 1893–94). Having no access to this journal, cannot verify the last reference.

THE PRESENT STATUS OF PSEUDODIPHTHERIA BACILLI.

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A committee was appointed in July, 1900, by the Massachusetts Association of Boards of Health to investigate the occurrence of diphtheria bacilli in well persons. It was found at the outset of their work that there existed such a wide divergence of opinion as to the identity of diphtheria-like bacilli that it became necessary, in collecting data, to make use of an arbitrary classification which included many doubtful types.

A glance backward will show that this uncertainty as to the precise status of diphtheria-like organisms has existed since the discovery of the specific cause of the disease. In his second communication on the etiology of diphtheria, Loeffler described the pseudodiphtheria bacillus as a non-pathogenic organism, interesting chiefly for its resemblance to B. diphtheriæ. Von Hofman, Vincent, Roux and Yersin, and others subsequently found it in the throats of healthy children and of those ill with measles, scarlet fever, nasal catarrh, and laryngitis. A controversy arose as to whether the organism was a distinct variety with no relation to B. diphtheriæ or a variant of it, lacking virulence, but in other respects identical. A few investigators took the latter view, notably Roux and Yersin, who supported it in a paper published in 1890. They defended this position with much force, pointing out that less difference existed between diphtheria bacilli and pseudodiphtheria bacilli than between virulent and attenuated anthrax bacilli obtained from the same source.

At the present time bacteriologists are divided into three groups by their opinions on this question.

1. Perhaps the greater number in this country believe that the term pseudodiphtheria bacilli should be applied only to that group of non-virulent organisms which resemble diphtheria bacilli very closely, but which may be distinguished from them by certain cultural or morphological characteristics. They regard diphtheria bacilli which produce toxin and those which do not as variants of the same organism.

2. A lesser number of bacteriologists in America and many Continental observers, including Roux and Yersin and Behring, believe in the identity of diphtheria bacilli and the so-called pseudodiphtheria bacilli, and regard the latter solely as varieties of B. diphtheriæ. Dr. F. F. Wesbrook, of Minnesota, divided all diphtheria bacilli into granular, barred, and solid forms, according to their appearance when grown on blood serum and stained with Loeffler's methylene blue. These forms he has further divided into types with reference to their shape and size.

The drawings which he presented with his descriptions leave no doubt that he considers several forms often classed as pseudodiphtheria bacilli undoubted diphtheria bacilli. His classification was that adopted by the committee of the Massachusetts Association of Boards of Health in the investigation previously referred to.

3. A few observers still apply the name pseudodiphtheria bacilli to those which are identical with diphtheria bacilli, with the single exception of being non-virulent, as well as to those which show cultural differences.

As might be expected, many methods have been devised to separate true diphtheria bacilli from those resembling them. A few biological differences have been observed. In glucose bouillon one distinction was found by some to be constant. All virulent diphtheria bacilli and some non-virulent ones are said to produce acid when grown in this medium, while pseudodiphtheria bacilli either produce no acid or produce it after a few hours' growth, and then rapidly produce alkali. The experience of different observers has differed greatly as to this, and it has been pointed out that their technique has been too dissimilar for a fair comparison of their results, and that, moreover, avirulent diphtheria bacilli cannot in many cases be distinguished from pseudodiphtheria bacilli by this means.

Neisser's method of staining the polar granules was hailed as a certain means of identifying true diphtheria bacilli, but it has been shown that not all virulent diphtheria bacilli possess polar granules, and that many avirulent diphtheria bacilli are without them, staining solidly. Furthermore, some pseudodiphtheria bacilli, even when grown less than twenty hours, have polar granules which stain beautifully by Neisser's method. Pioskowski's method is open to the same objection.

It has been noticed that these polar bodies develop more rapidly in true diphtheria bacilli than in the bacilli which most resemble them. The character of the emulsion resulting from mixing a colony with a few drops of water and several other apparently distinctive features have been described, but they have not been found equally reliable by different experimenters. And so we are obliged to say to-day that there are no methods of identifying true diphtheria bacilli which are regarded as trustworthy by all those who have devoted special attention to the subject.

The matter is not one of importance to laboratory workers alone, but has serious bearing on the control of diphtheria. One of the most vexing questions which confronts public health officers is the proper disposal of well persons infected with diphtheria bacilli, and this uncertainty about a matter so fundamental complicates it exceedingly. At present there is little difficulty in isolating persons who have had diphtheria until the bacilli can no longer be found in cultures from their throats.

In some cities a more or less successful attempt is being made to detain or throw some safeguards about those who have been directly exposed to the disease and who have infected throats, but no one has yet ventured to suggest that those well persons who carry diphtheria bacilli and who have not been directly exposed to the disease should be detained until their throats are free from infection. If we are to believe that all the so-called pseudodiphtheria bacilli are variants of true diphtheria bacilli, we must regard so large a part of the population infected that it is unfair to detain the few whom our bacteriological examinations detect, and it may even be held by some, as it is said to be by Behring, that it is unjustifiable to isolate even those suffering from the disease. It may be urged that the non-virulent forms may be disregarded, but even if it were practicable to test all cultures for virulence, animal inoculations afford us no positive assurance that, transferred to the throat of a non-resisting person, the bacilli might not cause disease. On the other hand, if such well-defined differences are observed in the growth or morphology of the organisms that it may be demonstrated that they constitute two or more distinct varieties, bearing no relation to each other, the problem of dealing with persons infected with B. diphtheriæ will be immensely simplified. It will then be entirely practicable to isolate all well persons in infected families who bear the germs and to compel others to take reasonable precautions against spreading the disease.

The matter is of so much importance that every effort should be made by bacteriologists to meet upon common ground in classifying diphtheria-like bacilli. Reports on cultural peculiarities should be so complete that one who has used somewhat different methods, and thereby failed to arrive at a similar result, should not be at liberty to deny the validity of another's conclusions.

As none doubts that if the organisms are identical all varieties must have had their origin in cases of clinical diphtheria, investigations should be made in places where diphtheria has not recently prevailed. Too many such examinations have been made in cities where diphtheria is endemic, and in jails, schools, and asylums, and too few in those country hamlets remote from railways, where, as far as the memory of the oldest physician serves, no case of diphtheria has occurred.

Careful observation of the morphology of diphtheria-like bacilli will probably be of greater value than a study of cultural differences. Dr. Wesbrook's classification, if enlarged to include a few types which cannot be conveniently grouped with any of his, forms a most admirable basis for comparison, and the water-colored drawings which accompanied his original article may well serve as models of accuracy.

In examinations made for the diagnosis of diphtheria it is rarely that the decision depends upon differentiating diphtheria bacilli and pseudodiphtheria bacilli; but when we are called upon to determine the

presence of diphtheria bacilli in well persons the difficulty is often insurmountable.

It is because any data, however fragmentary, which bear upon an important subject have a certain value that I present a few observations on the morphology of a single variety of pseudodiphtheria bacillus.

Diphtheria has prevailed at the Willard State Hospital since 1899. In the management of the epidemic it is considered of much importance to isolate all well persons among the inmates in whose throats or noses diphtheria bacilli may be found. To this end as great a number of cultures as the facilities of the laboratory permit are taken from the 2700 employés and patients residing in the institution. In 17,509 cultures from 1423 well persons which I have examined during the last year, undoubted diphtheria bacilli were found in 189 cases. The cultures were all grown overnight on blood serum. The large number of cultures does not permit plating each "suspicious" one or close study on different media, and so it has been necessary to adopt some rather arbitrary method of deciding which organisms found are diphtheria bacilli and which are not. As absolute uniformity in the conditions of growths exists, this plan has been followed by more success than might be imagined. In all cases in which considerable doubt is felt, another culture is taken and some special methods of growth employed, but in the vast majority of cases the decision is made from the morphology of the organisms present in the first cover-slip preparation.

The fact which, after this number of examinations, seems most prominent is that there is one variety at least of the diphtheria-like bacilli which possesses a constancy of form and distribution which entitles it to separate consideration. To avoid confusing it with a form to which several names have been applied, it may be termed, for the moment, "type a."

Occurrence. It has been found at the Willard State Hospital so frequently in the nose that it has come to be regarded as a normal inhabitant of that part. In a series of 100 cultures from the nose it is very common to find it in 25, and sometimes in 60, in most cases predominating in numbers over all other bacteria present. In a few cases I have found it in cultures in which were undoubted diphtheria bacilli, but all of these were from well persons, and I have not yet seen it in cultures taken from those suffering with diphtheria. It has been found in the throats of those in whose nose cultures it was present the same day and on other days, and, in somewhat less than 1 per cent. of all cultures examined, it has been found only in the throat. In many instances a culture from the nose shows no other organism; in a large number of cultures, staphylococci and "type a" are present together. In certain persons this organism is so constantly present that in cultures taken months apart it is possible to predict the finding with certainty,

while many individuals have had cultures taken repeatedly and "type a" never found.

VIRULENCE. I have inoculated only a few guinea-pigs and no other laboratory animals with cultures of this bacillus. None of the animals inoculated with pure cultures which died showed evidences of illness or of local reaction.

MORPHOLOGY. This bacillus shows less variation in size, shape, and staining than any other of the non-pathogenic bacteria found in the throat or nose. Whether the culture be from the nose, or the throat the morphology is the same. It is a moderately thick bacillus usually composed of two deeply stained segments separated by an unstained portion. The entire bacillus commonly forms an elongated ellipse. The segments are joined base to base, and each is spade-shaped rather than lancet-shaped. The segments are apt to be equal in length, although often one is longer than the other. When this is the case the shorter one is usually thicker than the other. The unstained portion is

FIG. 1.

FIG. 2.

just perpendicular to the long axis of the bacillus, and narrow and uniform in width. In some cultures about every eighth or tenth bacillus does not present this segmented appearance, but forms an evenly stained ellipse. In cultures which have grown longer than eighteen hours there is a tendency for the ends to take the stain a trifle more intensely than other portions, but after twelve or fifteen hours the bacillus stains deeply and evenly with Loeffler's alkaline methylene blue. The arrangement is not quite as irregular as that of B. diphtheriæ, four or five bacilli often arranging themselves like the spokes of a wheel. Fig. 1 shows "type a" drawn from a cover-slip preparation. It is exceedingly common to find diphtheria bacilli, even in initial cultures from clinical cases, which resemble this bacillus closely, and yet, in the greater number of such instances there is enough difference in the morphology to distinguish between them. When such bacilli occur in pure cultures of B. diphtheriæ they resemble the forms shown in Fig. 2, which is drawn to a smaller scale. The inequality in the two segments is greater, the

ends are more pointed, the sides are more concave, and the stain is rarely taken as evenly as by "type a."

This is not a complete description of the morphology of this type, but a few observations concerning a bacillus well known, often encountered, and variously named. If it can be generally agreed that this is a distinct variety, bearing no relation to B. diphtheriæ, and a distinctive name is given it, a great deal will be gained in classifying diphtheria-like bacilli, for it is certain that this organism is responsible for more difference of opinion in the examination of cultures from well persons than any other of the pseudodiphtheria bacilli.

A REPORT OF THREE MEDICOLEGAL CASES INVOLVING THE DIAGNOSIS OF CHRONIC DELUSIONAL INSANITY.[1]

By Sanger Brown, M.D.,
of Chicago.

My present purpose is not to discuss *in extenso* chronic delusional insanity, but merely to report briefly three medicolegal cases encountered in practice during the past year, in which the diagnosis mainly involved a consideration of that particular form of alienation, and, finally, to enunciate certain diagnostic criteria deducible from the histories presented.

Case I.—B. C. (seen at Detention Hospital), aged forty-two years, married, coal-dealer; correct habits, general health and family history good; came to Chicago from Germany at the age of fourteen, and soon after twenty started in business successfully for himself and proposed marriage to a former schoolmate, who accepted his offer, coming from the old country to consummate the ceremony. The union was a fruitful and happy one. Both were active and consistent church members, their clergyman often citing theirs as the model family of his rather large congregation.

About two years prior to the date of my visit B. C. returned home, not by design, but quite unexpectedly, however, and found D. alone in the family bedroom with his wife; the children all on the floor below by order of the mother, and she sitting on the bed, which had the appearance of having been used; D.'s clothing was disarranged. Now, D. had gone to school in the old country with both B. C. and his wife, and had accompanied the latter on her voyage to this country, and B. C. had given him work some years before, which threw the suspected parties much together, as B. C.'s residence stood in the yard where D. was employed. B. C. said he could have killed D. then and there, but refrained from making a scene on account of his family. D. never came to the house again, though he had formerly been a frequent visitor there.

[1] Also sometimes designated monomania, paranoia, and reasoning insanity. Read at the Illinois State Medical Association, April 29, 1903.

After this B. C. frequently upbraided his wife for her frailty, but did not speak of it to a third party until over a year later, when on the occasion of a social gathering at their home he chanced to discover her in the arms of F. in a stairway, whereupon he shortly afterward confided his trouble to his clergyman, and subsequently the wife confessed. All was peaceful until a few days later, when she told B. C. that the confession was a bogus one, and that she made it at the instigation of his mother and the clergyman in order to placate B. C.'s "fixed idea." This the clergyman denied *in toto*, at least in so far as his connivance was concerned.

Now, B. C. became more demonstrative than ever, at times loudly taunting and denouncing his wife before the children, even insinuating to her that one of them, differing in appearance from the others, was the offspring of D., and that possibly their illicit relations had begun as they voyaged together to this country, and he likewise recalled incidents which he thought gave support to this hypothesis. Finally, his fellow-deacons tried to heal the breach, but without success. The congregation divided on the subject, and B. C. was summarily arrested while at work and taken to the Detention Hospital.

The hearing in court developed no material facts in addition to those stated. I felt warranted in testifying that the charge of insanity had not been sustained,[1] because, even though B. C.'s conclusions might have been unwarranted, his accusations were based wholly upon and never extended beyond the two incidents above named. No evidence was forthcoming—and I sought carefully for it—of any vague or general suspicions either before or after these occurrences. B. C. was acquitted, has since lived apart from his wife, but contributes fairly to the support of her and their children, and attends to business as formerly; but his opinion regarding his wife's infidelity remains unchanged.

Case II.—G. H (seen at a private sanitarium, where he was in custody pending trial), aged fifty-four years, married, practical mechanic, inventor, and manufacturer; correct habits, general health and family history good. Married happily in early life, and has three married children. Though they have lived in amity, his wife states that she has always had to reckon with his excessive jealousy in her social relations.

Two or three months prior to the date of my visit G. H. conceived the idea that his wife was practising masturbation, observed her narrowly, and thought her attitudes and movements, especially in bed, proved his suspicions; would lie awake in an adjoining room at night listening; secured a powerful flashlight, which he turned upon her frequently while she slept, in order to take her unawares and define accurately her position. Finally, he told her he was certain she was a confirmed and almost constant masturbator, and begged her to go to a sanitarium for treatment.

Several weeks were consumed up to this point, at which time he likewise confided his suspicions to one of his sons-in-law, and added he was certain his wife was also having sexual intercourse with her house dog. Matters went on thus till about a week previous to my visit, when, at their summer home, where she was staying with her children, he accused his wife of criminal intimacy with K., a gentleman neither he nor she had ever met, and cited certain noises he heard about the house at night as confirmation of the charge. He shortly told his son-in-law he

[1] This diagnosis may seem to contradict the title of the paper.

was certain by the way his wife looked at men generally and the way her glances were returned that her sexual irregularities and marital infidelity were commonly known; he was greatly exasperated when his confidant ridiculed his stated convictions.

Finally, a day or two later, the family having assembled in the sitting-room, he appeared at the door with a loaded pistol in each hand and opened fire, saying excitedly that he would "compel them to listen to him." He did not appear to be actuated by anger against his wife, and did not aim at her particularly. Fortunately, he was overpowered before anyone was hurt, though one of the bullets passed through the sleeve of a member of the group. After his arrest, which immediately followed, he made several conflicting statements regarding his purpose in shooting. To me he admitted it was very foolish, but firmly maintained the correctness of the convictions above expressed.

Obviously here the delusive conceptions and conclusions germinated and flourished altogether upon a pathological activity of certain cerebral neurones or essential cerebral elements. In other words, they rested entirely upon a subjective basis; hence a diagnosis of insanity might be confidently pronounced.

Incidently, pending the legal proceedings, G. H. had a large and painful hemorrhoid removed—he had been troubled with piles more or less for many years—and on recovering from the anæsthesia his delusions appeared to have vanished. He apologized to his wife, who was present, and three weeks subsequently the legal proceedings were dismissed, no recurrence of his delusive ideas having been manifested in the meantime.

Case III.—L. P. (appeared in court as defendant in a charge of insanity preferred by the members of his immediate family), aged fifty-four years, merchant, married, three grown children; habits correct, general health and family history good, except that two maternal uncles became insane after middle life.

In his early twenties he married a charming and estimable young lady of distinguished family, and lived happily with her until about three years prior to the above date, when the shadow about to be described first fell across their path.

L. P. developed the idea, without any alteration of conduct on her part, that his wife was unfaithful to him; that she kissed her brother-in-law, a venerable clergyman, in a suggestive manner and quite unlike she had done before; and that further she lodged him in their house on an occasion when he paid them a visit, as he frequently did, with a view to having illicit sexual intercourse with him; that generally she admitted men surreptitiously and indiscriminately to the house for the same purpose, even specifying mechanics who were making repairs; and that his daughter, a girl of eighteen, and his mother-in-law, a lady nearly eighty, who lived in his home, were cognizant of his wife's transgressions, and aided and abetted her in them. After he had pledged them to secrecy, the coachman and housemaid were offered a large sum if they could furnish conclusive evidence of his conviction, and he showed them how to spy unobserved into certain rooms where he suspected his wife habitually participated in illicit sexual orgies. Later he confided his conviction to his grown son, and asked him to carefully watch the rear of the house at night to see if men—no particular man—did not surreptitiously enter. He smelled his wife's menstrual napkins to determine whether or not he

could determine an odor of semen upon them, claiming that he could do so—thus to his own mind conclusively confirming his suspicions. In company with his wife they met a mutual friend at a golf club, and he knew by the glances exchanged that they would remain at the club-house and cohabit while he was out on the course playing.

Afterward he sent a telegram to a favorite relative living a thousand miles distant to meet him half-way, as he had something of vital importance to communicate. At the meeting he related the above and much more of a similar nature as facts about which there could be no doubt. His relative accompanied him home, and, with the co-operation of others, succeeded in getting him to retract his charges, become reconciled to his wife, and take a trip for his health. This partial remission of his delusions occurred about eight months from the date of their first appearance. He returned home after about two months' absence, having, while away, however, occasionally thought he was being watched by detectives. A few weeks after his return his delusions recurred with full force, and continued up to the time of the trial. In the meantime he made several extended trips abroad, at times thinking he was in imminent danger of arrest and incarceration, sometimes even disappearing and concealing his whereabouts for several weeks together. Finally, shortly before the hearing, he ordered the proper allowance he had formerly made his wife reduced to a mere pittance, and stipulated further that she might only receive this on condition that she take up her residence outside the city limits, asserting that her conduct justified him in forcing this humiliation upon her.

Many business acquaintances testified that they had known L. P. for many years; had met him during the period covered by this investigation, and had never seen anything in him indicating insanity. And, indeed, it may here be stated that in none of the three cases under discussion was there anything in the demeanor, deportment, or general conversation suggestive of mental derangement. To the casual observer there was no noticeable impairment of judgment or of any of the mental faculties. Hence it not infrequently happens that a petition for the application of legal restraint to such patients is denied by a jury, which thus, perhaps, unwittingly, permits the perpetration of a terrible but preventable tragedy.

For the reasons stated in the preceding case I had no hesitation in testifying that L. P. was insane, and, further, that his expressed attitude relative to the reduction of his wife's allowance afforded ample ground for apprehending that he might make a homicidal attempt upon any of the parties concerned in his morbid convictions. The jury disagreed, and, after a few weeks, L. P. in the meantime having made a division of his income satisfactory to his family, the legal proceedings were dropped. He, however, still retains his delusions as before.

The essential diagnostic problem in these cases was whether the particular ideas in question were conceived and elaborated on a basis of objective data, denoting cerebral reactions not inconsistent with those natural to the individual, or whether they were wholly or essentially subjective, the product of autogenous pathological cerebral activity. In either case the conclusions reached might be ever so erroneous—delusions, indeed; only in the latter, however, could they properly be desig-

nated as insane delusions. If these criteria are kept clearly in view, the difficulty of reaching a correct diagnosis in these cases may, I think, be to some extent simplified.

Though Cases II. and III. are clearly illustrative of the inception and early course of chronic delusional insanity, and happen to exhibit many singular similarities, it would be erroneous to infer that they represent the most usual type of that disorder. It commonly commences in early life, even during adolescence, and, while the delusions frequently involve some phase of the sexual sphere, this is by no means constant. Indeed, the range and strength of the delusions present almost infinite variations when a large number of cases is examined. All, however, and I think this may be applied to insanity generally, exhibit in common the criterion cited above. That is, to repeat, the delusions rest upon a subjective basis, represent a morbidly autogenous manifestation of cerebral energy. In fact, the cerebral disturbance may be so great as to give rise to hallucinations of the special senses, morbid activity of the cortical centres related to the organs of special sense being erroneously accepted by the patient as the normal exercise of these organs.

The character of the delusions and the temperament of the individual in which they occur determine his conduct. Some of the subjects of this disorder, after having learned by experience that assertion of their peculiar convictions means deprivation of liberty, are able to repress them and live at large, sometimes even conducting an extensive business successfully; while others comprise the most dangerously homicidal patients ever encountered in the wards of a hospital for the insane.

Though subject to some fluctuations, or even complete remissions in its early stages, when the disease is once fairly seated permanent recovery is exceedingly rare.

MERCURIAL NEPHRITIS.*

By John M. Swan, M.D.,

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(From the Chemical Laboratory, Medical Department, University of Pennsylvania.)

In the concluding paragraph on the subject of mercury, H. C. Wood[6] says that "it appears to be established that certain disagreeable and perchance serious effects may be produced by mercurials when freely and continuously used in the treatment of syphilis, against which the practitioner must be on his guard. The most important of these is nephritis, with its consequent albuminuria."

* Read before the College of Physicians, Philadelphia, November 4, 1903.

The case which serves as the basis of this study illustrates in a strik ing manner the serious results of the overadministration of mercury:

The patient is a widow, aged thirty-one years.

Family History. Her father is living, at the age of seventy-four years; he complains of symptoms that point to the disturbances due to the uric acid diathesis, and recently has shown the early indications of posterior spinal sclerosis; at the age of thirty years he had an attack of psoriasis. Her mother is living and suffers from chronic gastric disturbance. One brother died of cholera infantum. Two sisters are living and in good health. One of these sisters has four daughters, each of whom suffers from periodic attacks of psoriasis. Her paternal grandfather died at the age of forty-two years, of cerebral hemorrhage; her paternal grandmother died at the age of eighty-seven years, of an unknown cause. Her maternal grandfather died at the age of fifty-two years, of cerebral hemorrhage; her maternal grandmother died at the age of thirty-seven years, of enteritis.

In April, 1898, the patient noticed an eruption, which first appeared on her arms and face, but which disappeared under treatment in about four months. In February, 1900, a similar eruption appeared, first on her face, whence it spread until it involved her entire body. At that time the eruption was associated with gastric pain and she was put on a restricted diet, so that in six months the eruption disappeared. A similar eruption appeared again in February, 1901. The eruption, at this date, first made its appearance on her face and then spread until it involved her entire body. The eruption persisted until October, 1901, at which time she consulted a dermatologist in another city, who told her that it was due to syphilis. She was then put upon a mixture containing $\frac{1}{32}$ grain of mercury biniodide and 15 grains of potassium iodide, which she took three times a day. In addition to this she took $\frac{1}{4}$ grain of the yellow iodide of mercury four times a day, and 2 drachms of mercurial ointment by inunction daily. The first prescription she used during October and November; the others she used during October, November, December, and January. During the time she was on this treatment she complained of soreness of the teeth, salivation, running at the nose, pains in the legs and in the abdomen, nausea without vomiting, and occipital headaches. Her bowels were loose and she passed two semisolid, fetid stools daily. The pains in her legs were cramp-like and deep-seated; they were paroxysmal in character and worse at night. These symptoms came on about four weeks after treatment was begun, which was then stopped for a week. At the end of this time the symptoms had disappeared and she began to take the medicines again and continued taking them for about three weeks longer, until the same symptoms reappeared. The administration of the medicines was again stopped until the symptoms disappeared, after which she was told to begin taking them a third time. On February 23, 1902, she began to complain of œdema of the feet, legs, and thighs, of occipital headache, and of pain in her eyes when they were exposed to the light. At that time she noticed that she was frequently passing a small quantity of urine at short intervals. About one week later she began to complain of dyspnœa and precordial pain. This pain was stationary, and was increased by movement and by deep inspira-

tion. In January, 1902, she was confined to bed for a week on account of universal pains, suppression of urine, and vomiting.

On March 2, 1902, the patient returned to her home in Philadelphia on account of dyspnœa, pain in the precordium, œdema of the lower extremities, and partial suppression of urine. She then had an eruption of small, scaly patches, most marked on the back; but also visible on the thorax and abdomen, the arms, the legs, and the face. Her pulse was 96 per minute, small, weak, but regular. Her heart action was rapid, the sounds were deficient in muscular quality, but no murmurs could be detected. Her urine was acid in reaction; had a specific gravity of 1014, and contained 1.4 per cent. of albumin by the Esbach method. Microscopically, the urine was found to contain large numbers of hyaline casts, renal epithelium, and leukocytes. The examination of the other organs showed no evidences of disease.

On account of the fact that a dermatologist in another city had diagnosed the skin eruption of which the patient complained as syphilis, I thought that the renal condition was due to syphilis. Cases of syphilitic nephritis have been reported, particularly one recently by Hoffmann and Salkowski,[1] which was cured by mercurial inunctions; and one reported by Mühlig,[14] which was cured by hypodermic injections of mercuric chloride. Hoffmann and Salkowski have no doubt that the syphilis caused the nephritis in their patient, which was indicated by urine containing hyaline casts and 7 per cent. of albumin. They also believe that the mercury cured the kidney condition; in fact, their patient, five months later, had a return of his syphilitic eruption and albuminuria with casts, which was again apparently cured by mixed treatment. Waldvogel[3] has also recently reported a case of nephritis due to syphilis which was cured by mercurial inunctions. This author, however, points out the difficulties in demonstrating absolutely that nephritis is due to syphilis in a given case.

In 1901 Ferras[18] published a thesis entitled "Studies on the Nutrition in Syphilitics by the Chemical Analysis of the Urine." Out of nineteen cases studied in preparing this thesis, one patient presented a serious albuminuria complicating a malignant syphilis. This patient passed 3300 c.c. of urine in twenty-four hours, which contained 16 gm. of albumin. After an exclusive milk diet for eight days it was found that the albumin remained stationary, that the general condition of the patient was not ameliorated, and that the urine had diminished to 1500 c.c. The author, therefore, concluded that the nephritis was dependent upon the syphilis and not on some cause existing before the syphilis was acquired. In spite of mercurial treatment, the albumin excreted increased to 40 gm. in twenty-four hours and the patient died of uræmia.

Believing that my patient was suffering from a nephritis due to the syphilitic poison, I put her upon treatment with ¼ grain of the yellow

iodide of mercury three times a day, increasing 1 grain daily; in conjunction with 2 drachms of infusion of digitalis and 1 drop of spirits of glonoin every four hours. After taking the yellow iodide of mercury for twenty-four hours the patient began to complain of sleeplessness and nausea without vomiting. At that time she was passing from 2000 to 2500 c.c. of urine in twenty-four hours, which had a specific gravity of 1021, and which contained hyaline and granular casts, renal epithelium, and leukocytes, and 0.7 per cent. of albumin by the Esbach method. The administration of digitalis was discontinued, but the mercury was continued until she had been taking it for five days. As the symptoms did not improve, but rather seemed to be aggravated, the administration of mercury was stopped. At that time she was taking 2 grains of the yellow iodide of mercury daily. After an interval of a week, treatment with mercurial inunctions was begun, 1 drachm daily, and after three days this was stopped because the patient complained of nausea, insomnia, occipital headache, foul breath, œdema of the feet, the legs, and the thighs. The eruption during this mercurial treatment showed no signs of improvement. The feet, the legs, and the thighs were painful as well as œdematous. There was a red line on the gums and the tongue was thickly coated. It then appeared plain that the eruption was not due to syphilis, and, in consultation with Dr. Jay F. Schamberg, a diagnosis of psoriasis was made. During this time the patient passed from 1500 to 2750 c.c. of urine in twenty-four hours, which contained from 0.35 per cent. to 0.6 per cent. of albumin, as determined by the Esbach method, as well as hyaline and granular casts.

It now appeared as though the nephritis from which the patient was suffering was due to the large quantities of mercury that she had taken. Her urine was examined for mercury according to the method of Almén, as given by v. Jaksch.[2] To 300 c.c. of urine a little sodium hydroxide and some saccharose were added and the mixture boiled. After boiling, the precipitate was allowed to settle and the fluid was decanted. The precipitate was then dissolved in hydrochloric acid and the resulting solution was diluted with six volumes of water. A small piece of thin, polished copper-foil was then placed in the fluid, which was maintained at a moderate heat for an hour and a half. The copper-foil was then removed, boiled in alkaline water, washed with alcohol and ether, dried and heated in a Reinsch tube to sublime the mercury. By this process mercury was obtained as small globules, the finding being confirmed by Dr. John Marshall.

This process of detecting mercury in the urine is very tedious, requiring about three hours, and Dr. Marshall suggested the following method as being simpler and more rapid, requiring only about one hour to complete the process. To 600 c.c. of urine 100 c.c. of strong hydrochloric acid are added, together with 0.5 gm. of potassium chlorate.

The mixture is heated to the boiling-point in a porcelain dish, and is then, while hot, transferred to a funnel provided with a cock in which a folded filter has previously been placed. The end of the funnel is placed in the wide portion of a thistle tube, the end of which has been fused so that it should have a small outlet, into which at the outlet a piece of bright copper-foil, rolled in the shape of a spiral, has previously been placed. The rate of passage of the hot filtrate through the thistle tube is regulated by the stop-cock in the funnel so that about 100 c.c. pass over the copper-foil in about seventy seconds. The liquid which has passed through the thistle tube is collected in a suitable receptacle, kept hot, and while hot passed over the copper-foil repeatedly until the entire amount of the fluid has passed over the copper-foil six times. The copper-foil is then washed in water, alcohol, and ether, and, when dried in the air, is heated in a Reinsch tube to sublime the mercury. This method was first applied on April 2, 1902, mercury having been detected in the urine by the process of Almén on April 1, 1902, and the patient's urine since then has been examined at intervals, first of one month, and later of two weeks. Mercury was found in all the specimens examined until April 16, 1903. From April 2, 1903, until April 16, 1903, the amounts obtained by sublimation in the Reinsch tube were very small. The last dose of mercury, 1 drachm of mercurial ointment, was administered to the patient on March 18, 1902. The patient had been eliminating mercury, therefore, for one year and twenty-nine days since the last dose was administered.

The œdema from which the patient suffered was deep-seated and, over the internal surface of the tibia, appeared to be situated beneath the periosteum. On one occasion I examined the urine for albumose, thinking some subacute inflammatory lesion of the bones might be present which would cause this substance to appear in the urine, but no albumose was found.

During the course of the treatment the patient was given hot salt-water baths, with the hope of increasing the elimination of mercury through the sweat glands. This produced a profuse red rash, somewhat similar to the rash of scarlet fever, and, as the lesions of psoriasis were also still present, the latter eruption formed circular, pale areas in the midst of the diffuse redness. As Hoffmann[12] reported two cases of mercurial dermatitis in which eosinophilia was present in the blood, a blood examination made during the persistence of this eruption gave the following result: erythrocytes, 4,840,000; leukocytes, 11,200; hæmoglobin, 100 per cent. Differential count: polymorphonuclears, 65 per cent.; lymphocytes, 24 per cent.; transitionals, 6 per cent.; eosinophiles, 5 per cent. It will be noted that there was a slight eosinophilia. Further, the granular degeneration of the erythrocyte, described by White and Pepper[13] in chronic lead poisoning, was looked for, but not found.

During the progress of the case the patient had several acute exacerbations, which were characterized by a diminished excretion of urine, increase of œdema, nausea and vomiting, and headache. Rest in bed, hot baths, and liquid diet controlled these in a day or two.

On July 1, 1903, the patient's condition was as follows: She had been teaching school for seven months, part of the time both day and night school. She feels tired and complains of pains around her heart, particularly at night; she is a little short of breath at times, usually in the evening. Her vision is good. There is probably slight œdema of the ankles in the morning. She has headache occasionally, when she is unusually tired. Her appetite is good, her bowels are regular, and she sleeps fairly well; little noises keep her awake, but if she is not disturbed she sleeps all night. She passes about 1500 c.c. of urine daily; an examination of the morning specimen gave the following result: color, amber; reaction, acid; specific gravity, 1010; sediment, slight; microscopic examination—few granular casts, large quantities of epithelium, leukocytes; chemical examination—albumin, 0.025 per cent. by the Esbach method; no sugar. She does not complain of palpitation, of nausea or vomiting, nor of indigestion. Her psoriasis is worse than it has ever been.

Her eyes are prominent; the pupils react both to light and distance. The mucous membranes are of good color. The tongue is clean and moist. Pulse 90 per minute, regular, small volume, artery normal.

Heart. Apex neither visible nor palpable. Dulness third interspace, midsternal line, midclavicular line, fifth interspace. The sounds are heard most distinctly in the fifth interspace just outside the midclavicular line. There are no murmurs. The muscular quality of the systolic sound is deficient. The diastolic sound at the base is accentuated.

The lungs are normal except for cog-wheel inspiration in the left axilla. The thyroid body is distinctly palpable.

Blood Examination. Erythrocytes, 4,500,000; leukocytes, 10,800; ratio, 1:416. Hæmoglobin, 82 per cent. Differential count: polymorphonuclears, 70.6 per cent.; lymphocytes, 24.6 per cent.; transitionals, 3.2 per cent.; eosinophiles, 1.6 per cent. There was no granular degeneration of the red cells.

She is taking ten drops of tincture of digitalis and one glass of lithia water daily. On account of having just got her system rid of one metallic poison, I did not feel warranted in giving her arsenic for the psoriasis, so put her upon 5-grain doses of potassium acetate three times a day.

Dr. Marshall's procedure for the detection of mercury in the urine is based on the theory that mercury is eliminated in the urine in some organic combination. The addition of hydrochloric acid and potassium chlorate to the urine breaks up the combination and converts the mercury into a soluble chloride. Metallic mercury is then deposited from the hot acid solution on the copper-foil by electrolysis, so that, by sublimation in the Reinsch tube, it redeposits on the walls of the glass tube and can be detected as small globules adhering to the walls

of the capillary portion of the tube, with a low power of the microscope. When Dr. Marshall suggested the process he thought it was original, but subsequently he found essentially the same process described by Witz,[5] except that Witz used potassium permanganate instead of potassium chlorate to assist in the decomposition of the organic mercurial compound.

The accumulation of mercury in the tissues, its subsequent elimination, and its possible deleterious effects are subjects of considerable importance to the physician, particularly so to the genito-urinary specialist and the syphilographer. White and Martin[7] point out that lesions of the kidney particularly predispose to the development of hydrargyrism and say that the drug should be used with caution in cases in which there are crippled kidneys.

V. Jaksch[20] says that when the diagnosis has been correctly made the administration of mercury in syphilis has never done permanent evil, although individuals that are afflicted with nephritis tolerate the drug badly. The urine in such cases contains albumin, casts, and renal epithelium. Cornil[21] says that mercury has a profound action on the kidneys. It is, in the first place, an irritant to these organs and causes a swelling with fatty degeneration of the epithelium of the tubules.

According to Wood,[6] mercury has been found in the blood, the urine, the serum of ulcers, the saliva, the feces, the pus from ulcers, the semen, the milk of nursing women, and in every conceivable secretion and in every tissue. It has been demonstrated that a single dose of mercury does not remain in the system, but when the drug is administered constantly for a length of time, elimination does not keep pace with absorption, so that the drug accumulates in the tissues. There appears to be no limit of time during which stored-up mercury may remain in the body. All the probabilities point to the possibility of mercury being deposited in the tissues in such form that it is practically inert and exerts no effect on the system; liable, however, under certain agencies to be set free and exert its power upon the general nutrition. Wood refers to Klemperer's conclusions* that the changes in the kidneys due to mercury are: excessive hyperæmia, parenchymatous nephritis, hemorrhagic nephritis, widespread degeneration of the epithelium, and deposits of chalky material. My patient has apparently reached the condition of parenchymatous nephritis with widespread degeneration of the renal epithelium, which, I fear, is permanent.

Hare[4] says that there are three theories concerning the absorption of mercury: 1. The theory of Miahl, that the mercurial preparations are transformed in the stomach and intestines into the bichloride, which, in turn, unites with the sodium chloride in the blood and circu-

* Virchow's Archiv, cxviii., 1889.

lates as a double chloride of mercury and sodium. 2. Henoch's theory that the metal forms an albuminate of mercury. 3. Voit's theory that a chloro-albuminate is formed. Hare also says that mercury is eliminated as an albuminate by every excretion of the body. A single dose is rapidly eliminated, but large doses frequently repeated show a cumulative action, and the mercury is so slowly eliminated as to remain for indefinite periods. He refers to the work of Balzer and Klumpke, which shows that mercury can be eliminated by the kidneys for many weeks when the body is saturated with the drug in doses of $\frac{1}{16}$ grain daily.

The literature, besides that referred to by Wood and Hare, contains a few reports of cases of nephritis due to the ingestion of mercury and of the finding of mercury in the urine. These cases are usually instances of poisoning by a single large dose of some preparation of the metal. For instance, Benham and Hendly[8] report the case of a girl, aged twenty years, who took ninety grains of red oxide of mercury and ammoniated mercury. After the symptoms of acute poisoning had passed off the urine became scanty and contained a large amount of albumin and casts. By the test with a piece of bright copper-foil, mercury was found in the urine, saliva, and the stools. The patient presented a vivid-red rash, like the rash of scarlet fever. The girl recovered after a miscarriage, but there is no note concerning the ultimate condition of the kidneys.

Axenfeld[9] reports the case of a man who presented the symptoms of chronic mercurial poisoning from using acid nitrate of mercury in the preparation of rabbit skins. The urine of the patient was normal in quantity and did not contain albumin. A quantity of urine, evaporated to a small volume, was decolorized with a hypochlorite and a portion then treated with hydrogen sulphide; a black precipitate of mercuric sulphide was produced. Another portion of the liquid treated with potassium iodide gave a red precipitate of mercuric iodide. Mercury was also obtained as a whitish-gray deposit on a strip of gold wrapped around a layer of tin (Smithson's pile) from another portion of the liquid. Mercury was found in the saliva by the same reaction.

Seguin[10] reports the case of a boy who took red iodide of mercury and potassium iodide for a cerebellar tumor. The symptoms of the disease improved under the treatment; but on examining the urine it was found to contain hyaline and epithelial casts, but no albumin. The administration of mercury was stopped and the renal condition improved. Then the symptoms of cerebellar tumor returned and mercury and potassium iodide were again given. This time the urine contained albumin, hyaline and granular casts, and free renal epithelium. He also notes a case the particulars of which were given to him by

Kinnicutt. The patient was taking mercury biniodide for secondary syphilis. He also had a urethritis. The urine contained albumin, leukocytes, and hyaline casts. The leukocytes and the albumin were due to the urethritis and disappeared with that lesion, but the casts persisted. The casts disappeared when the iodide was stopped, although the administration of mercury was continued.

Bouchard[14] reports the case of a man who was suffering from secondary syphilis and who had had an attack of lead poisoning eight months before. When first seen he had no albumin in his urine. After he had been treated with mercurial inunctions for five days he began to complain of swelling of his gums, and in spite of stopping the use of the mercurial ointment a sharp attack of stomatitis developed. Under the influence of this intoxication, seven days after the beginning of the stomatitis, the urine showed marked changes. The total quantity in twenty-four hours was 150 c.c.; specific gravity, 1017; albumin and traces of sugar were present, and the urea content was 3.6 gm. per litre.

Holle[15] quotes a fatal case of nephritis, reported by Riegel, in which the patient died from injury to the kidney and general poisoning after the prolonged administration of calomel. He also refers to a case reported by Pel in which the patient died soon after the administration of calomel, which had not produced diuresis as had been hoped. In this case it was found that there were large deposits of mercury in the kidneys.

Chauffard[16] reports the case of a young woman who took 5 gm. of mercuric chloride followed by 10 c.c. of laudanum. Among the symptoms referable to the kidneys in this case were complete anuria for five days, after which the patient began to pass 70 c.c. of albuminous urine in twenty-four hours containing an abundance of granulofatty casts and epithelial cells. The patient died from the effects of a complicating pericarditis twenty days after taking the poison. No mercury was found in the urine, and at the autopsy the kidneys were found to belong to the class of large white kidneys.

Chauffard and Gouraud[17] report a case of syphilitic nephritis in a man, aged forty-seven years, who had had scarlet fever at the age of six years. In spite of energetic mercurial treatment the patient died. At the autopsy it was found that both kidneys were swollen; the convoluted tubules were most affected and their lumina were filled with albuminous coagula. The glomeruli were not congested, but were surrounded by the same albuminous exudate, which, in certain places, half-filled the capsules of Bowman. The authors hold that the large quantity of albumin found in their case, 55 grams to the litre, argues in favor of the syphilitic origin of the nephritis, and say that this condition is quite the contrary in cases of mercurial nephritis. It seems to

me more probable that this was a case in which kidneys injured by scarlet fever were put into active inflammation by their efforts to eliminate the mercury administered for the treatment of the syphilitic infection than that it was one of syphilitic nephritis as claimed by the authors.

Cornil[19] reports the case of a hatmaker, who had been under the toxic influence of mercurial vapor for seven years. He died soon after he was admitted to the hospital suffering from clonic convulsions. He had neither alcoholism nor syphilis. At the autopsy a fatty degeneration of nearly all the striped muscles of the body was found. Mercury was found in the brain. The liver and the kidneys showed a fatty degeneration analogous to that observed in intoxication.

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THE CARDIOSPLANCHNIC PHENOMENON.

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To my knowledge this is a heretofore undescribed phenomenon as far as its clinical manifestations are concerned. The facts, however, up to the point of its clinical identification, have been fully established by the physiological investigations of others. These facts are correlated to intra-abdominal tension and the effects of such tension on the circula-

tion. It may be apposite before describing the phenomenon in question to succinctly review a few essential points on the subject of intra-abdominal tension. In the norm the latter is greater than the atmospheric pressure, and its positive pressure is exerted on the viscera, which in turn press on the abdominal parietes, causing them to bulge. Should a reverse condition of things prevail, the abdominal walls would become retracted. Positive intra-abdominal pressure is subject to two chief conditions, viz., atmospheric pressure upon the yielding abdominal walls and the contraction of the abdominal muscles. Therefore, intra-abdominal pressure is most pronounced in individuals with well-developed musculature and least evident in multiparous women with flaccid abdominal walls. Clinicians recognize the secondary effects of low intra-abdominal tension in conducing to splanchnoptosis, for with relaxation of the abdominal walls the support to the underlying tissues becomes defective and ptosis of the viscera is the likely result. The effects of reduced intra-abdominal tension on the circulation has received but scant consideration from clinicians—a most lamentable fact, considering the gravity of the issues involved.

Hall and Barnard[1] have contributed exhaustively to this important subject which has been practically elaborated in the excellent work of Campbell.[2] They have demonstrated that there is a tendency of the blood to accumulate in the splanchnic area, with consequent syncope. Like the generality of veins, the great splanchnic veins are very susceptible to pressure, and the amount of blood within them is greatly influenced by pressure on the abdominal walls. Mere pressure of the latter suffices to squeeze out of them a large quantity of blood. Thus, gravity, posture, the accoutrements of dress and other facts greatly influence the amount of blood contained in the splanchnic area. More blood accumulates in the splanchnic veins in the erect than in the recumbent posture, and it is not an uncommon observation for syncope to occur in bedridden patients who are suddenly constrained to leave the bed. The removal of stays in women often induces a feeling of faintness, and the same symptoms may occur when a large quantity of ascitic fluid is removed and in susceptible individuals when the bladder is emptied or feces discharged. There are some subjects who experience a sensation of faintness even in health when much pressure is brought to bear by the abdominal muscles in the evacuation of the bowels, and it is not an uncommon observation to note, when the patient is very ill, that the administration of a purgative may be followed by symptoms of collapse. In all these instances there is a determination of blood to the splanchnic area with consequent brain anæmia. The sensation of faintness or syncope is much accentuated when the abdominal walls are flaccid. Three factors enter into consideration in the mechanism of blood supply to the splanchnic vessels, viz.:

(1) contraction of the abdominal muscles, (2) respiration, and (3) the regulating vasomotor action of the splanchnic vessels. The first factor is an important one, the transversales maintaining the anterior and posterior abdominal walls in fairly close contact, preventing in the erect posture the gravitation of blood to the splanchnic area. The second factor concerns the diaphragmatic descent and pulmonary suction. Each time the diaphragm descends the intra-abdominal vessels are compressed, and the action thus exerted is less evident in the firm arteries than in the flaccid veins, the blood being squeezed from the latter into the right heart. De Jager[3] has shown that even strong pressure upon the abdomen has little or no effect on the arteries, but serves to express a large amount of blood from the splanchnic veins. Hill has also shown that in consequence of some failure in certain compensatory mechanisms the blood gravitates into the splanchnic veins from the right heart, and that abdominal compression will send the blood back from the veins to the right heart, thus re-establishing the circulation. Such compression not only augments the input into the right heart, but likewise increases, according to some observers, the systemic arterial pressure by increasing the peripheral resistance in the splanchnic area. Pulmonary suction refers to the large quantity of blood drawn into the lungs with each inspiration, and this physiological process has not been inaptly compared to a species of dry cupping. Chapman[4] avers "that if at the termination of expiration the quantity of blood in the lungs is from $\frac{1}{15}$ to $\frac{1}{18}$ of the total quantity of blood in the body, at the termination of inspiration it will be from $\frac{1}{12}$ to $\frac{1}{18}$." The pulmonary vessels expand with each inspiration and contract during expiration, the results being an increased flow of blood from the right heart and the lungs; the dilated vessels, as Campbell[5] puts it, "actually suck the blood out of the right heart." The final factor, the splanchnic vasomotor mechanism, in preventing the gravitation of blood into the splanchnic veins, is an important one. Hill and Barnard[6] have demonstrated that the splanchnic vasomotor mechanism suffices to combat this contingency, but when this mechanism is inhibited, as occurs when the splanchnic nerves are cut, a second mechanism is brought into prominence, viz., expiratory compressions of the abdomen occurring simultaneously with inspiratory thoracic suctions, the former squeezing and the latter sucking the blood out of the splanchnic pool. The latter mechanism compensates ineffectually in carrying on the circulation, and is referred to by Campbell[7] as the "respiratory mechanism." That the latter is not as efficient as the vasomotor mechanism is evidenced by the fact that the effects of gravity may be entirely compensated after the injection of curare, which paralyzes the muscles. Both mechanisms may be inhibited by division of the spinal cord at the first dorsal vertebra, and if the animal operated on be held with

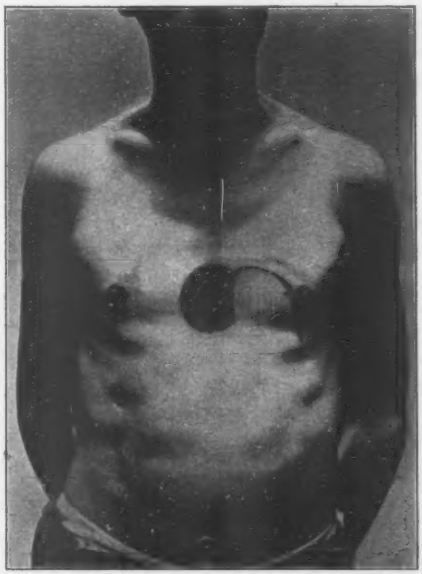
the head up all the blood accumulates in the splanchnic veins and the empty heart ceases to beat; if, however, the abdomen of the animal is compressed, the blood is expressed into the heart and the circulation is restored. With the foregoing facts at our command, we will be able to appreciate what I have called the cardiosplanchnic phenomenon. If the lower sternal region—*i. e.*, the sternum contiguous to the heart—is first percussed in the standing and then again in the recumbent posture, one may appreciate a decided alteration in the percussion tone; in the former attitude it is resonant or even hyperresonant, in the latter it is dull or flat. This is the cardiosplanchnic phenomenon. It may be elicited, but less effectively, when the patient is in bed and sits up. Vigorous compression of the abdomen will exaggerate the phenomenon in all instances. Not infrequently the relative cardiac dulness will extend beyond the right border of the sternum. In no instance was this percussional phenomenon absent, although hundreds of individuals were examined. In a few instances the phenomenon was only feebly expressed by mere attitudinal changes, yet reinforcement by abdominal compression made its elicitation easy. A few forced inspirations at once dispelled the phenomenon, and it could likewise be made to disappear by the application of a vacuum cup to the abdomen. If the liver and spleen are percussed, first in the erect and then in the recumbent posture, a decided variation in regional percussion is obtained. In the former attitude the area of dulness is increased; in the latter it is diminished, and the splenic area of normal dulness may completely disappear. Abdominal compression exaggerates the results just as it does in increasing the sternal dulness. These conditions are superadded to the cardiosplanchnic phenomenon. Primarily it is necessary to adjust the discrepancy of my observations with those already accepted, and then to analyze the manifestations which give genesis to the phenomenon. Theoretically, one could assume at the outset that the situs of the heart more nearly approaches the anterior chest wall in the erect than in the recumbent posture, and, indeed, it is not only assumed but accepted by clinicians, that in the latter posture the heart falls away from the thoracic wall. Kingscote[8] perpetuates a similar error by assuming that the predominance of asthmatic seizures at night was caused by the posture of recumbency, the dilated heart in this position causing it to impinge on the vagi. I have shown elsewhere[9] the fallacy of this contention. With the aid of the Roentgen rays the triangular spaces in front and behind the heart are clearly defined in forced inspiration, and the rays give undeniable demonstration that change of posture only slightly modifies, if at all, the spaces in question; in fact, it may be observed that in not a few instances the anterior triangle is diminished and the posterior triangle is increased in recumbency. The foregoing facts contravene the contention that

posture influences the relation of the heart to the chest wall. Again, percussional results are often influenced by the prejudiced preconceived ideas of the clinician. The skilled clinicist in his interpretation of percussional phenomena is guided not so much by the ear as by the *tactus eruditus;* thus, palpatory percussion will often yield results which would wholly escape the observer who is influenced only by what he hears and not by what he feels. In the elicitation of the cardiosplanchnic phenomenon, palpatory percussion must be our chief mentor. Others may contend that my lung reflexes (that of contraction and dilatation) and heart reflex may account for the phenomenon in question, but these reflexes have been assiduously eliminated and play no rôle in the cardiosplanchnic phenomenon. It is true that in recumbency the splenic and hepatic areas of dulness become diminished; this is in accordance with the well-known fact that postural changes influence the position of the lung borders, the so-called passive mobility of the lung borders. In recumbency the lower lung border descends about one-half inch lower than in the erect posture. The decrease in hepatic and splenic dulness as associated signs of the cardiosplanchnic phenomenon takes into consideration only the deep or relative hepatic dulness, together with the additional fact that the areas under consideration are diminished to an extent not to be accounted for by mere passive mobility of the lung borders. The cardiosplanchnic phenomenon permits of simple explanation. In the erect posture the blood leaves the right heart, which topographically is beneath the lower sternum, and tends to accumulate in the splanchnic area, whereas recumbency opposes this influence of gravity. Abdominal compression, which in some instances must be vigorous, assists still further in expressing the blood from the splanchnic veins and sending it back to the heart. The same factors prevail with relation to the liver and the spleen, the quantity of blood in the latter viscera being in direct proportion to the amount of blood contained in the splanchnic veins, and in inverse proportion to the amount of blood contained in the heart. Forced inspirations hasten the output of blood from the heart, hence the almost immediate evanescence of the cardiosplanchnic phenomenon after vigorous breathing. To those who seek the elicitation of the phenomenon care must be bestowed on the fact to counsel the subject to conduct superficial breathing, but not to suspend it, for otherwise this manœuvre alone will cause the blood to accumulate in the right heart. Exhaustion of air by means of a vacuum cup to the abdomen will decrease intra-abdominal tension and increase in consequence the quantity of blood in the splanchnic veins. After the latter manœuvre, the right heart chambers will become depleted. I have endeavored by means of the Roentgen rays to determine whether any of the manœuvres already suggested for provoking the cardiosplanchnic phe-

nomenon in any way influence the cardiac diameters, and my investigations show that they do not. If the latter observation is correct, how then are we able to account for the increased dulness of the lower sternum, the necessary concomitant of the cardiosplanchnic phenomenon. The sternum contiguous to the right cardiac ventricle in the norm yields percussional resonance which is caused in the main by the transmission of the percussion blow to the neighboring lung tissue. A percussion blow is propagated from one and one-half to two and one-half inches on the surface, and to a depth of about two and one-half inches. It is evident, then, that in the normal subject the dulness of the right ventricle is not sufficiently pronounced to dampen the sound obtained from the vibration of air within the lung alveoli. It is, however, possible to conceive that if the right ventricle were sufficiently filled or even overfilled with blood—and this is capable of fulfilment without any increase in the dimensions of the cardiac chambers—the normal resonant sound of the lower sternal region would become dull or even flat. Our final endeavor is to show the value of the phenomenon in diagnosis and treatment. In estimating the size of the liver or spleen by percussion we are constrained to regard three factors, viz., position of the patient, the amount of blood in the right heart, and the vigor of respiration. The liver size is normally dependent, in part on the amount of blood which it contains, and this has been estimated by Foster[10] to be equivalent to one-quarter the amount of blood contained in the body. The skilled diagnostician will not find it a difficult task to demonstrate reduction in hepatic and splenic dulness after repeated forced inspirations. Such reduction cannot wholly be accounted for by the opening up of atelectatic lung areas, for in the case of the liver my investigations only refer to the deep or relative hepatic dulness. Supposing the object of our examination is an enlarged liver or spleen, and that we desire to know how much of the enlargement is due to hyperæmia and how much to tissue hyperplasia? Percussion in different attitudes, repeated inspirations, and abdominal compression, if necessary, will solve this question. If a crucial test is to be applied, all that is necessary is to apply a vacuum cup to the abdomen, and then, in accordance with the facts already established, the liver and spleen would still further show augmentation in size by percussion. Of course, each observer must estimate for himself to what degree the normal liver or spleen will enlarge under such conditions.* Supposing a patient complains of sternal pressure, which, if dependent on a

* Since writing the above I have directed attention to the "liver reflex" (Occidental Medical Times, September, 1903). This reflex is readily available for diagnostic purposes. It consists essentially of retraction of the lower liver border from a half-inch or more as determined by percussion, after irritation of the skin contiguous to the lower liver border. The liver reflex is pronounced in enlargement of the liver dependent on hyperæmia, and slight or absent, if due to connective-tissue hyperplasia.

dilated heart, would disappear on application of a vacuum cup to the abdomen, the blood would be expressed from the heart to the abdomen and the sensation of pressure would disappear at once. Let us further assume that we are confronted with an abdominal neoplasm and the question arises, are we dealing with a growth connected with the liver or spleen, the application of the vacuum cup may solve the problem, for the latter viscera would show augmentation in size and conversely they would show diminished size were the abdomen compressed. The elicitation of the cardiosplanchnic phenomenon would prove valuable



The cardiosplanchnic phenomenon. The shaded area is the circumscribed patch of dulness elicited after vigorous compression of the abdomen; the contignous area is the superficial or absolute cardiac dulness.

in the differential diagnosis of a dilated heart from a pericardial exudate; in the latter affection it would not be elicited. Idiopathic syncope and vertigo and the vertiginous attacks of Glénard's disease may be attributed to a defective splanchnic vasomotor mechanism. Now, in the average normal subject the cardiosplanchnic phenomenon is fairly evident, but in the conditions just cited it would be exaggerated. In other words, the more perfect the mechanism, the less pronounced the phenomenon. A large number of respiratory affections owe their dyspnœa to an overtaxed right heart, and this is notably the case in asthma. While I do not agree with Kingscote,[11] that a dilated heart

is the invariable concomitant of asthma, yet I do contend that an enlargement of that viscus is operative in predisposing to a paroxysm and augmenting its severity. In a severe asthmatic attack which resisted the conventional remedies, a vacuum cup to the abdomen arrested the attack. In this instance, suggestion probably played no rôle, insomuch as the relief of the paroxysm coincided with the disappearance of substernal dulness.

It is unnecessary to adopt the theory of Kingscote that a dilated heart striking the vagi is responsible for nocturnal exacerbations of asthmatic paroxysms, for an overburdened right heart is a sufficient explanation. Indeed, the predominance of dyspnœic attacks at night in cardiorespiratory affections can be explained by the augmented blood supply to the right ventricle, the mere result of recumbency. In my opinion the upright posture instinctively assumed in orthopnœa is not only due to the fact that the extraordinary muscles of respiration may work to better advantage, but also for the additional reason that the blood from the right heart is enabled to gravitate to the splanchnic veins. In dyspnœa from any cause the implication of the heart in this symptom may be gauged by expressing blood from the right heart by means of the vacuum cup applied to the abdomen; if the dyspnœa is relieved we have reasonable assurance that it is caused by an overburdened heart. Many other instances could be cited to show the importance of this phenomenon in diagnosis, but the examples already mentioned will suffice. In treatment, the phenomenon suggests many possibilities in the direction of re-establishing the circulation and in relieving the heart when it is overtaxed. While the estimation of blood tension is a reliable gauge in estimating the value of any therapeutic method which has for its object cardiac stimulation, yet we cannot deny the fact that cardiac activity may be awakened without any corresponding increase in blood tension, just as we may awaken the respiratory centre by cutaneous stimulation without any necessary increase beyond the normal in the number and depth of the respirations.

My own observations show that the elicitation of the cardiosplanchnic phenomenon is unattended by any increase in the blood tension. Our only guide in estimating the amount of blood which is diverted to the right heart is the cardiosplanchnic phenomenon, not only the phenomenon *per se*, but the degree of its demonstration. The precordial dulness is, as has already been intimated, not so much dependent on the approximation of the heart to the chest wall, but rather to the amount of blood contained in the cardiac chambers. The following manœuvres in order of citation will express the largest amount of blood to the right heart: 1. Suspension by the feet, a method easy of execution in children. 2. Inversion of the patient. 3. Compression of the abdomen. 4. Recumbency. Inversion or suspension of the subject by the feet accentuates the dulness to the left of the lower sternal

region and diminishes it over the latter area. In syncope it appears to me that the object achieved is not so much the determination of blood to the anæmic brain as it is to the determination of blood to the heart. To effect compression of the abdomen in acute conditions demanding cardiac stimulation, paroxysmal abdominal compression will suffice; in chronic conditions a firm cushion applied to the abdomen and secured by a rubber bandage or broad strap which permits of any degree of traction will prove serviceable. The objection to continuous abdominal compression is the respiratory embarrassment often experienced by the patient. An overtaxed right heart is present in so many cardiorespiratory conditions that it may be apposite to select croupous pneumonia as a paradigm. There are so many indications and contraindications cited by writers for venesection in this affection that it is really problematic whether bleeding is beneficial or harmful; at any rate, it is an empirical practice. It appears to me that in pneumonia our object should be to preserve the quantity of blood, for it is the chief stimulus for maintaining the cardiopulmonary circulation and furnishes the leukocytes, which do so much in conducing to a favorable issue. Instances do occur when bleeding is justifiable, and it would prove more effective if the patient were bled into his own vessels. The application of a vacuum cup to the abdomen meets the emergency and should be employed, if only as a tentative measure, should an overburdened heart with its consecutive phenomena warrant its employment. If this manœuvre is effective, it may be repeated any number of times, as it is a method harmless and painless if the exhaustion of the cup is not executed beyond a reasonable degree. It may be remarked in conclusion that alterations in blood tension do not synchronize with the intensity of the cardiosplanchnic phenomenon. This may be explained by the fact that the great amount of blood which is expressed into the right heart makes the latter unable for the time being to disburden itself of this large access of blood. The condition thus induced is somewhat similar to that when the breath is held, and Marey has shown, by tracings of the right ventricle when breathing is suspended, that its beats become slower and that it manifests an increasing difficulty in emptying itself, the amount of residual blood increasing with every beat.

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THE ETIOLOGY, PATHOLOGY, AND DIAGNOSIS OF ADRENAL HEMORRHAGE.

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In this paper I have recorded four cases of hemorrhage into the adrenal glands, and also entered into a short discussion on the subject.

Case I. *Thrombosis of the superior longitudinal sinus and the cerebral and cerebellar veins; pin-point hemorrhages in the cerebellum; extensive purpura; hemorrhage into the left adrenal gland and left kidney; thymic abscess; staphylococcus aureus and albus cultivated from the heart blood.*—Annie M., aged two years and eight months, admitted December 12, 1902, under the care of Dr. Eustace Smith, to the East London Hospital for Children.

The child was obviously rickety, and is said to have been delicate from birth. There was no evidence of congenital syphilis. She had been vaccinated successfully.

The present illness commenced on December 3, 1902, with pain in the head and general weakness. On December 6th a "swelling" was noticed at the angle of the jaw, on the left side. The child had frequent screaming fits, but no true convulsions.

On admission to the hospital the infant appeared to be very ill. She lay in an apathetic state, but occasionally put her hands to her head and screamed, as if she suffered great pain. Temperature was 101° F. The veins of the scalp were greatly distended, especially on the right side, and there was also a very prominent vein about the middle of the forehead. A large abscess was found on the left side of the neck, and a smaller abscess was situated in the lower part of the left pectoral region. The knee-jerks were brisk. Kernig's sign was absent. There was double otorrhœa. There was nothing else abnormal noted in any other part of the body.

December 13th. Infant was worse. The face was a pale leaden color. There was slight cyanosis. Temperature was 103° F., and the child was semicomatose. Respiration was rapid. Pulse was rapid and running. A small patch of purpura appeared on the outer side of the left knee, and gradually spread over the rest of the limb. Toward the end of the day the right foot became similarly affected, and the purpura gradually spread upward. There was swelling of the limbs but no pitting on pressure. There were no convulsions. Death occurred on December 14th.

I performed the post-mortem examination sixteen hours after death. Nutrition was fairly good. There was a large abscess on the left side of the neck beneath the lower jaw, which contained about half an ounce of thick yellow pus. There was no communication between this abscess and the pharynx. Another small abscess was found in the seventh intercostal space which contained about a drachm of pus. This had no connection with the ribs, thorax, or abdomen. It was situated

between the skin and the chest muscles. There was deep-red staining of both legs and considerable swelling of the right thigh; but when one cut into these tissues they appeared to be normal. There were no abnormal changes found in the femoral artery or vein. All the thoracic viscera were normal, except the thymus gland, which was enlarged, owing to an abscess in the right lobe, which contained thick yellow pus.* The left kidney contained about a dozen "black spots," which were situated in the cortex, and were found to be hemorrhagic. Capsule stripped readily. No other naked-eye changes observed. Right kidney appeared to be normal. Bloodvessels were normal.

Left Adrenal Gland. This was simply a "coffee-colored" hemorrhagic mass, which was quite obvious through the capsule of the gland. There appeared, however, to be a small covering of apparently normal cortex, which formed a wall for the blood clot lying in the medulla. I regret that I failed to notice the condition of the suprarenal vein. There were no hemorrhages into the surrounding tissues.

Right adrenal gland was apparently normal. Mesenteric glands were normal.

Brain. There was complete thrombosis of the superior longitudinal sinus, the superior and middle cerebral veins, and a slight extension of the clot into the right lateral sinus. The cerebellar veins were also completely thrombosed. The clot was tough, pale red, and firmly adherent to the vessel walls. Pinpoint hemorrhages were visible in the gray matter of the cerebellum, but none elsewhere. There was no evidence of meningitis, but some œdema of the pia mater. There was no excess of fluid in the ventricles. There was softening of the brain in the region of the thrombosed vessels. Cranial bones were normal. Mastoids were normal.

Bacteriology. A pure culture of the staphylococcus aureus was obtained from both subcutaneous abscesses; and both the staphylococcus aureus and albus from the blood of the right ventricle.

Microscopic Examination. There were numerous scattered hemorrhages all through the medulla of the left adrenal gland, and in some places the hemorrhages occupied quite a large area of the gland tissue. The capillaries were overdistended, and in many places the walls of the bloodvessels had ruptured. Large, prominent, and distended capillaries were also seen between the columns of cortical cells, but otherwise the cortex was normal. The parenchyma of the medulla was unaffected except in the hemorrhagic areas. Here, many of the adrenal cells were destroyed, others had undergone "a granular degeneration"; a few apparently consisted of little more than a nucleus. Sections of the gland were stained by Gram's and Pappenheim's methods for microorganisms, but with negative results.

The renal epithelium of both kidneys had undergone a considerable degree of cloudy swelling. Many of the glomeruli of the left kidneys were injected, and a few large scattered hemorrhages were seen in the cortical tissue.

The walls of the superior longitudinal sinus were injected and showed small-celled infiltration, while the lumen was filled with fibrin, and blood corpuscles, leukocytes, and endothelial cells.

The right adrenal gland was normal.

* Unfortunately no bacteriological examination was made.

CASE II. *Extensive burn; large hemorrhage into the right adrenal gland, associated with symptoms which rather pointed toward an acute lesion in the abdomen.*—Rose S., aged six years, admitted January 22, 1903, under the care of Mr. Keetley into the West London Hospital, Hammersmith. The child had burns of the first and second degree on her face, back, and both forearms. Temperature was 96° F. Boric acid fomentations were applied.

On January 24th the child was very restless and vomited during the night. Temperature was 99.8° F. January 26th she was restless and suffered from frequent vomiting. She is said to have complained of pain in the abdomen, but it moved well on respiration, and there was no localized tenderness. Pulse was 110. Temperature was 98° F.

January 27th. Abdomen moved well on respiration. Vomiting had stopped. Extremities were cold. Pulse was 140. Temperature was 98° F. Urine contained no albumin. Death occurred in the evening of January 27th.

Post-mortem examination was performed about twenty-four hours after death. There was very little found at the autopsy. All the thoracic organs were normal. The mesenteric glands were calcified. The right adrenal gland was very much swollen, of a deep-red color, and there was a very large hemorrhage into the medulla of the gland which so distended the cortex that it formed a sort of capsule. Left adrenal gland was normal. All the other abdominal viscera were normal. Brain was normal.

Microscopic Examination. Through the kindness of Dr. A. E. Russell, I was able to examine the suprarenal gland and to prepare specimens for microscopy.

The whole of the medulla of the gland was composed of blood clot. There was no parenchymatous tissue left. The cortex was very vascular and contained numerous vessels engorged with blood, but there were very few hemorrhages, and the parenchyma was apparently normal. No micro-organisms were seen in any of the specimens.

CASE III. *Gangrenous varicella; tubercular mesenteric glands; tubercular ulceration of the intestines; double adrenal hemorrhage; pure culture of the pneumococcus isolated from the heart blood and from the adrenal glands.*—Beatrice G., aged one year and ten months, admitted March 5, 1903, under the care of Dr. Coutts, to the East London Hospital for Children, with the following history:

Child had been restless, and had loss of appetite for about a fortnight before admission to the hospital. Typical varicella rash appeared on Monday, March 2d. Papules rapidly became vesicles, and as some of these spots subsided others appeared. On Wednesday, March 4th, some of the spots rapidly spread, and tended to form "large spreading sores." On March 5th the child vomited twice and was so ill that the mother brought her to the hospital.*

On admission. The trunk, head, and limbs were covered with bluish-black "ulcers" which varied greatly in size. The large ulcers were confined to the back. The floor of the ulcers was dry and glazed, and

* Three other children in the family had gangrenous varicella about the same period. Two recovered, but the third has since died in the East London Hospital from bronchopneumonia. The adrenals were healthy and there was no tuberculosis.

the edge was in some places serpiginous. Some of the small varicella spots were in the same condition. Several ulcers were present on the scalp. There were no purpuric spots. There were no physical signs in the lungs or other viscera. Pulse was extremely rapid, 200 per minute, and the respiration was also very rapid. Throat was normal. Child was almost comatose, and died within twenty-four hours of admission to the hospital.

Post-mortem examination was performed on March 7th. Body was well nourished. Skin covered with ulcers, as already described. There was well-marked evidence of rickets. Throat, larynx, trachea, and pharynx were normal. Pleuræ were covered with numerous hemorrhages, but there was no fluid in either sac.

Lungs. There were numerous dark-red hemorrhagic areas about the size of a pin's head, or a little larger, in the left lower lobe, otherwise there was no consolidation of either lung. Tracheal and bronchial glands were normal. Heart was normal. Epicardial and pericardial hemorrhages were found. Right lung was normal. Mesenteric glands were pale and about normal in size, and most of them contained small caseous masses.

Intestines. Lower end of the ileum and colon contained large tubercular ulcers, with localized tubercles on the peritoneal surface of the gut. There were no adhesions and no free fluid. Both adrenal glands were swollen and of deep purple color, but the bloodvessels were normal. There were no lesions elsewhere in the abdomen. Brain was normal. Bones were normal.

Bacteriology. A pure culture of the pneumococcus was isolated both from the heart blood and from the adrenal glands, but no confirmatory inoculation experiments were made.

Microscopic Examination of the Adrenal Glands. The whole of the parenchymatous tissue of the glands was almost entirely replaced by blood clot. Some of the cells of the zona glomerulosa and fasciculata appeared to be normal, but the great majority were destroyed. The capillaries were engorged with blood. No organisms were detected in any of the microscopic specimens.

CASE IV. *Bronchopneumonia; sudden death; double adrenal hemorrhage.*—Charles M., aged fourteen weeks, was seen by Dr. Jordan Harvey (to whom I am indebted for the notes of the case), on Thursday, January 23, 1903, just before death.

The child had been delicate from birth, otherwise there were no special points of interest in the case until the morning of the above date, when the mother noticed that the child's breathing was labored. The dyspnœa rapidly increased until death, which occurred at 11 P.M. on the same day. There had been no vomiting, diarrhœa, abdominal pain, convulsions, or any purpuric spots.

Post-mortem examination was performed by Dr. Jordan Harvey within twelve hours of death.

The body was pale and flabby, and weighed twelve and one-half pounds. The anterior fontanelle was greatly depressed. There was a patch of eczema over the left ear.

Chest. The pleural sacs were normal. The upper lobe of the right lung was solid from confluent patches of bronchopneumonia, and the lower lobe of the left lung was in a similar condition. The other thoracic organs were healthy.

Abdomen: Adrenal Glands. Both glands were deep purple in color and distinctly swollen. The cortex and medulla appeared to be equally affected. The bloodvessels were not examined. All the other abdominal organs were healthy. Brain was normal.

No bacteriological investigation was made.

Microscopic Examination. The whole of the medulla of the glands was composed of blood clot. A few scattered nuclei seen here and there in the various sections, and some cells which had no nuclei and stained in an irregular manner proved to be the only evidence of any parenchymatous tissue. The capillaries in both the medulla and cortex were greatly distended with blood.

Numerous isolated hemorrhages were seen in the cortical portion of the glands, but there was less destruction of the parenchyma than had occurred in the medulla. No abnormal changes were seen in the walls of the bloodvessels. No micro-organisms were found in any of the specially stained sections.

The Clinical Aspects of Hemorrhage into the Adrenal Glands.

Adrenal hemorrhage is generally a complication of various maladies, but occasionally there are symptoms which appear to be somewhat characteristic and lead to the diagnosis of a separate and possibly distinct disease. It has generally been found that other clinical phenomena have been sufficient to attract the entire attention of the observers, and the condition of the suprarenal glands has never been referred to.

As already stated, the clinical aspects are various, and have been considered from many standpoints by such writers on the subject as Arnaud, Still, Graham Little, and others. The first named has contributed the most valuable monograph on the subject, both from a clinical and a pathological standpoint. He gives the following classification: (1) the asthenic type; (2) peritoneal; (3) nervous. This classification is to my mind incomplete. The most interesting group is omitted. I should therefore add to Arnaud's classification:

1. Cases with a very acute onset and many symptoms which lead one to suspect an acute specific fever.

2. Cases with severe skin lesions, purpura, etc., but not suggesting an acute specific fever.

3. Those cases in which the adrenal hemorrhage occurs as a complication of some pre-existing disease, and is only recognized at the autopsy.

4. Hemorrhage in the newborn.

Possibly this classification is inadequate, but it will be found to meet the large majority of cases.

The most interesting group (No. 1 in my classification) has been fully dealt with by former writers on the subject. The chief points of interest

in these cases are briefly as follows: A very acute onset, generally in infants who previously are said to have been healthy. There are one or more convulsions, vomiting, diarrhœa, and within a few hours from the onset of the illness a petechial or purpuric eruption appears, which may spread diffusely all over the child. Pyrexia varies from 101° to 105° F. Collapse rapidly supervenes, and death takes place within twelve to forty-eight hours. The diagnosis of a specific fever has been made in many of these cases, and more especially variola, chiefly from the fact that the infants have generally been *unvaccinated*. Dr. Voelcker[1] describes a case in the *Pathological Reports of Middlesex Hospital for* 1894, of a well-nourished child, aged two years, who was admitted in November, 1894, into the Middlesex Hospital with a history of a severe illness of twenty-four hours' duration. The child was *unvaccinated*. There were vomiting, marked prostration; temperature 105° F., and an extensive petechial eruption which extended over the whole body. The case terminated fatally within forty-eight hours from the onset. It was recorded as doubtful variola. *Post-mortem* evidence showed hemorrhage into both suprarenal glands. Dr. Andrews[2] in the *Transactions of the Pathological Society of London*, reports a similar case to the above, in a well-nourished female child, aged one year and three months, who was *unvaccinated*, and where death also occurred within forty-eight hours. Both suprarenals were hemorrhagic. A. E. Garrod and J. H. Drysdale,[3] in the same *Transactions*, refer to the case of an *unvaccinated* female child, aged four months, who was brought to the hospital dead, covered with a blotchy purpuric eruption. Both adrenals were deep purple in color, and were found to be hemorrhagic. Dr. Batten,[4] also in the same *Transactions*, describes the case of a male child, aged two and a half years, which had been attending the out-patient department at Great Ormond Street Hospital for some time, suffering from lichen urticatus. The child was taken suddenly ill with vomiting and diarrhœa. Temperature was 102° F. Knee-jerks were exaggerated. Temperature rose to 106° F. before death, which occurred within forty-eight hours from the onset of the attack. The right adrenal was found to be hemorrhagic, but the left was normal. Eustace Talbot[5] reported two cases in the *St. Bartholomew's Hospital Reports* for 1900.

Case I.—A well-nourished child, aged five months, *unvaccinated*, was brought to the hospital suffering from severe abdominal pain, vomiting, and convulsions of a few hours' duration. Temperature was 101° F. There was no rash, but the child was not admitted, as it was thought that the case was probably infectious. About four hours later the mother returned to the hospital with the child moribund, and death rapidly supervened. *Post-mortem:* There was double suprarenal hemorrhage.

CASE II.—A well-nourished child, aged five months, *unvaccinated*, was suddenly seized with vomiting, abdominal pain, convulsions, and a temperature of 101° F. There was no rash. Death occurred within twenty-four hours from the onset of the illness. Double adrenal hemorrhage was found at the autopsy.

Clive Riviere*[8] recorded four cases in the *Transactions of the Pathological Society of London*. The children were all under the age of twelve months. *Three were unvaccinated*. One had been vaccinated and showed two good scars. There were vomiting, diarrhœa, and pyrexia, and death supervened within forty-eight hours; in one case within twelve hours. In every case there was hemorrhage into the suprarenal gland.

Graham Little,[7] in the *British Journal of Dermatology*, has referred to the case of a child, aged four months, which was admitted to St. Bartholomew's Hospital under the care of Sir Dyce Duckworth, suffering from a purpuric eruption all over the trunk and limbs. There was some pyrexia. The child was rickety and was *unvaccinated*. Death took place within twenty-four hours from the onset of the illness. Both suprarenal glands were found to be deep purple in color.

Another case of great interest is also quoted by Graham Little.[7] A male child, aged thirteen months, was admitted to the Royal Free Hospital, under the care of Dr. Sainsbury. There was a diffuse purpuric eruption on the arms, legs, and face; also conjunctivitis, vomiting, convulsions, cyanosis, and pyrexia which reached 108° F. just before death, which took place within twenty-four hours from the commencement of the illness. There was a large hemorrhage in the right suprarenal gland. The left contained numerous hemorrhages.

This practically completes the list of these extremely interesting cases in which the diagnosis has caused such great difficulty.

What is the nature of these cases?

First and foremost, are they examples of fulminating smallpox? This diagnosis has been made in some cases, in others strongly suspected.

Fox,[8] in his article on hemorrhagic fulminating smallpox, says: "It runs its course precipitately and at times most unexpectedly, sometimes killing the patient in a few hours, in other cases not completing its career until the fourth or fifth day." There seems to be an entire absence from the literature of smallpox of cases similar to those recorded above, and at such an extremely early age. It is stated that infants are not so susceptible to smallpox as children and adults, but this is doubtful. Cases of fulminating smallpox often have extensive purpura, but present none of the characteristic features of variola, because death occurs at

* These cases have also been recorded by Graham Little in the British Journal of Dermatology, and by Blaker and Bailey in the British Medical Journal.

the commencement of the disease. Practically the only strong evidence in favor of smallpox in the cases under discussion rests on the fact that so frequently the children were unvaccinated (nine cases out of twelve recorded). It would be interesting to know, however, the exact percentage of *infants* who are brought to the various hospitals *unvaccinated*. There is nothing else in the clinical history of the cases which is not perfectly in accordance with an acute bacterial infection, fulminating scarlet fever, or measles, etc.

A most interesting case is recorded by Fox,[8] of an adult male, who died within twenty-four hours from the commencement of an illness which was considered to be hemorrhagic scarlet fever. This diagnosis was afterward found to be incorrect, as a friend who had occupied the same room developed smallpox within a fortnight. This is one of the few recorded cases in adults which resembles in many ways the cases described above, but this proved to be infectious, while every case I have collected failed to convey infection. This is most remarkable when we consider the overpopulated districts in which most of these children lived. MacCombie,[9] in his article on smallpox in Allbutt's *System of Medicine*, says: "I have not seen this fulminating form, which is characterized by its extreme rarity, rapid onset and termination, and by high temperature, delirium, coma, and collapse, rapidly terminating in death after a few hours' illness. No skin hemorrhages are seen, only visceral." In the recorded cases, however, skin hemorrhages have usually occurred. Lastly, physicians of such experience as Osler[10] and Immermann[11] do not refer to cases similar to those recorded in this paper in their various contributions to the literature of smallpox. It is extremely probable that we are dealing with an acute toxæmia of unknown origin, quite apart from any acute specific fever.

As already stated, the cause of the disease is unknown. In one case, however, the child had been given "some sausage" prior to the onset of the illness. This suggests the possibility of a gastrointestinal origin in some cases.

What Arnaud describes as the "peritoneal" type closely resembles the cases recorded above, except that the children were older, some had been vaccinated, and there was no purpura. In fact, in most examples of this class, the diagnosis has been made of an acute lesion in the abdomen.

About two years ago I was called to see a little boy, aged three years, who died with symptoms common to both types now under discussion. As far as I can remember the history was as follows: A woman took her two little boys for a day's excursion into the country. The mother and one of the children ate a pork pie on the journey home. Within a few hours both mother and son were seized with vomiting, diarrhœa,

and acute abdominal pain. When I first saw the child, about twelve hours after eating the pie, he had a high temperature (the vomiting and diarrhœa had now stopped), and he was covered with a diffuse purpuric eruption. Death supervened within twenty-four hours from the onset of the illness. A post-mortem examination was not allowed. The mother recovered after a long illness. The other little boy, who did not take any pie, was unaffected. Without the history of a gastrointestinal infection this case would no doubt be classed, by some people, among those resembling an acute specific fever. To my mind it serves to illustrate that we are not always dealing with one class of cases, but only with various clinical phenomena dependent on one main pathological lesion, adrenal hemorrhage.

A history of food poisoning only rarely presents itself in our cases. In ptomaine poisoning diffuse purpura appears to be a very rare occurrence, although visceral hemorrhages have frequently been recorded.

Goodhart and Still[15] report cases in infants of a week old, who have died quite suddenly with severe abdominal pain and collapse. At the autopsies hemorrhage has been found in one or both suprarenals, which have ruptured, allowing extravasation of blood into the surrounding tissues and peritoneal cavity.

Graham Little[7] has recorded cases which he has collected from the literature associated with purpura, but where the symptoms in no way resembled an acute specific fever.

Perhaps the most interesting case has been described by Arnaud.[12] A man, aged thirty-six years, was admitted to hospital in a state of coma. Cerebral hemorrhage was diagnosed, and all "other causes" were excluded, so far as it was possible to do so.

Post-mortem Examination: Both suprarenals were practically destroyed by an extensive hemorrhage into the gland tissue. All other organs were found to be normal. This case is the best example in the literature of adrenal hemorrhage presenting such symptoms that even in competent hands a diagnosis was made of a lesion of the central nervous system.

I can find no certain examples in the literature of the asthenic type of Arnaud, and cases which belong to class 4 (of my classification) will only be considered from a pathological standpoint.

Etiology. 1. In most cases the term "idiopathic" might justly be applied.

2. A gastrointestinal infection.

3. Very common occurrence in the newborn.

4. In both acute and chronic lung and cardiac disease, and in convulsions; in fact, any disease which is known to produce stagnation of

the blood in the veins or a marked increase of blood pressure may be associated with adrenal hemorrhage.

5. Septicæmia, pyæmia, acute miliary tuberculosis,* and the toxæmia from severe burns, etc.

6. Congenital syphilis. It would have been a matter of some surprise if congenital syphilis had been omitted from the list of causes, but there is very little to substantiate the opinion held by some observers as to the importance of this factor in adrenal hemorrhage.

7. Blows in the back, fracture of the spinal column, and severe abdominal lesions, such as ruptured liver, etc.

8. The so-called blood diseases—*e. g.*, scurvy, etc.

9. Lastly, adrenal hemorrhage is sometimes found at the autopsies on chronic diseases of the nervous system—*e. g.*, general paralysis of the insane,

Pathology. *Experimental Evidence on Animals.* Roux and Yersin,[14] by injecting diphtheria bacilli under the skin of rabbits, guinea-pigs, and pigeons, induced general dilatation of bloodvessels, congestion of the intestines and kidneys, and, almost constantly in guinea-pigs, congestion, and less often hemorrhage of the suprarenal glands.

Various other organisms appear to produce the same result—*e. g.*, Gaertner's bacillus, bacillus pyocyaneus, bacillus mucus capsulatus, pneumobacillus, streptococcus pyogenes, and the bacillus pestis, etc.

Bacteriology. Considerable importance has been placed by some observers on the fact that an organism has been found in the blood or tissues after death, and therefore that organism has produced the disease in question. To say the least, such a statement is likely to lead to a difference of opinion. In one of my cases the staphylococcus pyogenes aureus and albus were cultivated from the heart blood. In another case I obtained the pneumococcus in pure culture both from the heart blood and also from the suprarenal glands. Drysdale found the "streptococcus" in one of Talbot's cases. Riviere isolated the bacillus coli communis from the splenic blood and a saprophyte from the heart blood in one of his cases. Riesman found at the autopsies on six newborn children who had adrenal hemorrhage the staphylococcus aureus and albus in the spleen and right suprarenal of one case; and in three of the other cases he cultivated the staphylococcus albus and aureus from the adrenal glands. In every other recorded case of adrenal hemorrhage, from which a bacteriological examination was made, the results proved to be negative. It is possible that the streptococcus found in one of the cases, and the pneumococcus in

* Still reported a case of a male child, aged fourteen months, who died of acute miliary tuberculosis and hemorrhage into the left adrenal gland. There was no purpura.

another, may have had some important bearing on the causation of the disease, but the results are not by any means proof positive.

When we consider the numerous organisms which produce adrenal hemorrhage experimentally in animals, it is not unlikely that we shall find a parallel in the human subject.

Lately I have examined carefully the condition of the adrenal glands in several cases of bronchopneumonia and general pneumococcic infection in children, but have not found a single example of adrenal hemorrhage. Congestion might be described in one or two cases, but when we consider the delicate structure of the medulla of the suprarenal and the congestion likely to result from the mechanical effects due to consolidation of the lungs, absolutely no importance can be placed on such results. Again, a boy was admitted to the East London Hospital for Children with infective periostitis of the left radius and tibia, due to the staphylococcus aureus. At the autopsy pinpoint abscesses were found in practically every organ, and the staphylococcus aureus and albus were obtained from the heart blood, spleen, and kidneys. The suprarenal glands appeared to be fatty to the naked eye, otherwise normal. Microscopically two small hemorrhages were seen in one of the adrenal glands, and both glands would possibly be described by some writers as congested, but there was no fatty change. Undoubted adrenal hemorrhage was seen in two cases which died with marked cyanosis from cardiac disease, but this is probably a very common occurrence, and of no interest.

Two cases have been already reported of adrenal hemorrhage associated with burns. The first case was described by Churton,[16] of double adrenal hemorrhage which followed on a severe burn. The other, by Arnaud,[17] of a girl, aged seventeen years, who was found to have a large hemorrhage into the medulla of the right gland, but the left gland was normal. There was no bacteriological examination made in either case. The last-named writer also refers to two cases of liver abscess, one due to a suppuration of a hydatid cyst, the other post-dysenteric, associated with adrenal hemorrhage. In the latter case there was also phlebitis and thrombosis of the left suprarenal vein.

Morbid Anatomy. The following is probably the most suitable classification:

1. The whole gland or glands are converted into "blood sacs," with occasional extravasation into the surrounding tissues.*

2. A hemorrhage may occupy the medulla of the gland, but the cortex is spared, except for a few red corpuscles scattered between the columns of adrenal cells.*

* Thrombosis of the adrenal vein may be met with in either class of cases.

3. Scattered hemorrhages into the gland substance, chiefly in the medulla, with little destruction of the parenchyma.

I have not referred here to congestion of the adrenal glands. There is no doubt that it is very hard to decide what should be justly termed congestion. The medulla has such a rich blood supply, and the vessels are only, so to speak, blood spaces, that anything and everything may lead to what is termed congestion. It is quite certain that it occurs very often and is a very much commoner lesion than we are led to suppose. Arnaud[12] fully justifies my remarks; he says: "The frequency of vascular lesions of the suprarenals is difficult to estimate, as unless there is bronzing of the skin the examination of the capsules is generally neglected. It would certainly be found to be less rare if a systematic examination was made." In a hundred post-mortem examinations he found from personal observation three cases which showed large hemorrhages into the adrenal tissues; eight cases with smaller hemorrhages which were identified by the microscope, and lastly, hyperæmia and congestion in eighteen cases.

Post-mortem Appearances of the Adrenal Glands. They are either swollen and deep purple color, or there is one large hemorrhage visible to the naked eye, such as one sees in any other part of the body. In some cases the medulla is a very dark red and entirely composed of blood clot, while the cortex is apparently normal.

Thrombosis of the adrenal vein or veins has been noted in some cases, but it is important to examine the vessels in every positive or suspected case. This can be readily accomplished by putting the tissues on the stretch, when the bloodvessels will be plainly seen.

From the recorded cases the right gland appears to be more often affected than the left. Mattei[17] considers that this may be due to either of two causes: (1) that the right adrenal vein opens directly into the inferior vena cava, or (2) that the gland is compressed between the liver and the vertebral column.

Lastly, what is the relation of adrenal hemorrhage to Addison's disease?

When we come to think of the extremely high death rate which is probably associated with the former disease, it is unlikely that there are many cases of Addison's disease which are primarily due to a vascular lesion of the adrenal glands apart from tubercle. There is only one place where we are able to diagnose adrenal hemorrhage with any degree of certainty, viz., the post-mortem room.

Pigmentation is one of the cardinal signs of Addison's disease, and purpura is very commonly associated with adrenal hemorrhage; possibly purpura stands in the same relation to acute destruction of the adrenals, as pigmentation does to chronic. These two clinical phenomena seem

to me to be the most important points in the association of the two diseases.

Rolleston,[18] in Allbutt's *System of Medicine*, says: "Suprarenal hemorrhage has been associated with Addison's disease." Again, Hektoen[15] remarks: "The substitution of the blood clot by connective tissue might lead to induration and contraction, with atrophy of the medulla, which, if bilateral, might suspend the internal secretion and lead to Addison's disease." There may be a very slow destruction of the glands by hemorrhage, and, as a result, fibrosis, with the production of Addison's disease; but if this rarely does occur, it must be very rare. Everything tends to show that the large majority of cases of adrenal hemorrhage are very acute, and terminate fatally long before there is time for true Addison's disease to develop.

In conclusion, my best thanks are due to Drs. Eustace Smith and Coutts for the loan of their cases, and also to Mr. Keetley for permission to make use of the notes of the case which had been under his care.

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REVIEWS.

A System of Physiologic Therapeutics. Edited by Solomon Solis Cohen, A.M., M.D., Professor of Medicine and Therapeutics in the Philadelphia Polyclinic; Lecturer on Clinical Medicine at Jefferson Medical College; Physician to the Philadelphia Hospital and to the Rush Hospital for Consumptives, etc. Vol. IX. Hydrotherapy, Thermotherapy, Heliotherapy, and Phototherapy. Illustrated, pp. 570. Philadelphia: P. Blakiston's Son & Co., 1903.

This volume contains also special chapters on Balneology and Crounotherapy, notes on American Springs, Classification and Distribution of Mineral Waters in the United States, Saline Irrigations and Infusions, and on cognate subjects. The Physiologic Basis of Hydrotherapy in four chapters, with a brief exposition of the fundamental principles and practical applications of heliotherapy and phototherapy is from the pen of Professor Wilhelm Winternitz, than whom no one has enjoyed a larger experience with or greater enthusiasm for the use of water as a therapeutic agent. We may accept this as an exposition of his matured experience and as the strongest argument for the use of these physical agencies which can, at this time, be presented. While the practitioner of a broader experience may admire the thoroughness of his work in the study of the physiological facts, and may assent to their application in therapeutics, yet he may differ as to their applicability to the needs of daily practice. Moreover, he may find that other methods will more speedily, more pleasantly, and more surely yield better or at least equally good results. Should he reach this conclusion, then physical agencies will still find a place in his therapeutic equipment but as adjuvants. This position is still tenable and in consonance with a thorough appreciation of the valuable work which the author has done. To say that this section is the most forcible presentation of the subject, from the philosophical standpoint with which we are familiar, is but to give the author his just due. Dr. Alois Strasser presents the technique and methods of hydrotherapy in a thoroughly practical manner and with painstaking detail: a fitting sequence to Winternitz. Special hydrotherapy by Dr. B. Buxbaum considers symptoms and diseases, how these methods must be varied, and offers some statements as to the results which may be expected.

He is optimistic, as an advocate should be; dogmatic even to the point of compelling attention. To this section supplemental chapters by Dr. T. H. Kellogg on "Heliotherapy, Phototherapy, and Thermotherapy," and by Dr. Harvey Cushing on "Saline Infusions and Irrigations," are added. Of the first we may say that it is interesting, instructive, and, based on the great opportunities to be found in a large and favorably known institution, thoroughly practical. Of the last so much has been done in this line, both at home and abroad, that a thorough acquaintance with the literature, which is here shown, has entailed an enormous demand upon the author's time and patience. We believe that the physician and surgeon will find this the best presentation of the subject, and we have in mind the excellent work of both Kemp and Dawbarn.

"Balneology and Crounotherapy" is written by Professor E. Heinrich Kisch. This section has for its introductory chapter a dissertation on the "Classification of Mineral Waters," by Dr. Albert C. Peale, which leads to a more intelligent understanding of what may be anticipated and as well a broader view of this rather neglected subject. In order that resources at our doors may receive proper attention the services of the well-known writer, Dr. Guy Hinsdale, have been levied upon. The result of these combined efforts is in the highest degree satisfactory, and, whether in enumeration or in claimed efficiency, nothing to be desired is left for the most patriotic reader, and yet foreign-bath methods and drinking-cure plans are fully presented. If our nosological catalogue may seem unduly long, certainly the list of places and resources can rival it. In fact, this section compares favorably with works especially devoted to this particular subject. The translation is satisfactory, and to Dr. Augustus A. Eshner much credit is due. That such words as "explicable" (p. 149) have crept into the text is pardonable when the peculiarities of idiom are at times perplexing. In this volume the editor is more in evidence. First he very properly objects to the hybrid "Crounotherapy" and suggests "Loutrotherapy" as an improvement, and with this we agree. Secondly, in the Appendix he offers "Additional Methods of the Therapeutic Use of Water, Heat, Cold, Light, and Mineral Baths." This adds much to the value of the book, and could readily have been made still more indicative of what has been accomplished in this country in the way of simplification and adaptability. Others, whose work is not mentioned, have made important contributions, and even if their writings lack dogmatism their influence has been none the less important. Of volumes presenting much excellence we would single out this as one which has impressed itself upon us, not from the importance of the subject, but rather from the thoroughness with which the methods are illustrated and the ability shown in its presentation.

R. W. W.

HYPNOTISM: ITS HISTORY, PRACTICE, AND THEORY. By J. MILNE BRAMWELL, M.B., C.M. London: Grant Richards, 1903.

WHATEVER one's opinion concerning the usefulness of hypnotism may be, this is a very interesting book. It is, of course, written by a man biased in favor of his subject, but that is characteristic of the majority of the books written on the subject. Judicial calm is not to be expected, or at least is very rarely met with, in the discussion of a matter like hypnotism. Much less claim of its therapeutic value is made in this book than in many others. The historical chapter is very entertaining and contains quite a number of facts not mentioned in other historical sketches. For example, it is not commonly known that Herbert Spencer published in *The Zoist* essays "On the Situation of the Organ of Amativeness" and "A Theory Concerning the Organ of Wonder." This, to be sure, was a long time ago, and proves only that even geniuses may sometimes jump the wrong way. In Bramwell's opinion, "the evidence as to the production of blistering and changes of temperature by suggestion is by no means conclusive." All experiments on clairvoyance that he has seen he regards as valueless, an opinion in which most sober-minded men will agree with him. He has found nervous, ill-balanced, and hysterical people the most difficult to influence by hypnotism; healthy people who possessed the power of mental concentration the easiest. He thinks that apparent hypnosis in animals is often if not always really a willed simulation of death for purposes of protection. He believes the greatest therapeutic use of hypnotism is in functional nervous diseases. There is a quite long chapter on the theories of hypnotism. Everyone interested in the subject should read this book. C. W. B.

PROGRESSIVE MEDICINE. A Quarterly Digest of Advances, Discoveries, and Improvements in the Medical and Surgical Sciences. Edited by HOBART AMORY HARE, M.D.; assisted by H. R. M. LANDIS, M.D. Vol. III., September, 1903: Diseases of the Thorax and its Viscera, including the Heart, Lungs, and Bloodvessels; Dermatology and Syphilis; Diseases of the Nervous System; Obstetrics. Philadelphia and New York: Lea Brothers & Co., 1903.

THIS issue of this valuable quarterly contains four most important sections, namely, those on Diseases of the Thorax and its Viscera, including the Heart, Lungs, and Bloodvessels, by William Ewart, M.D., F.R.C.P.; that on Dermatology and Syphilis, by William S. Gottheil, M.D.; on Diseases of the Nervous System, by William G. Spiller, M.D., and on Obstetrics, by Richard C. Norris, M.D. The

volume, as usual, is very much more than a mere year book of medical literature. Those in charge of its various departments are qualified to write *ex cathedra* on the subjects committed to them. Instead of taking all the current articles as they appear, and making abstracts of them, the editors confine their attention to the most important contributions which have appeared during the year, and discuss them in such a way as to present what is really valuable in them to the reader in an available form. There is no medical publication which has more thoroughly earned the respect and confidence of the medical profession, and which more thoroughly deserves its support than this periodical, which has so long maintained its pre-eminence among all the innumerable year books, epitomes, and annuals, which have recently endeavored to compete with it.

These four volumes practically constitute a text-book of medicine and surgery, appearing revised up to date each year. They are not only invaluable for reference to those who wish to write, but they furnish the practising physician with information on all subjects upon which he may desire a guide. J. H. G.

DISEASE OF THE PANCREAS: ITS CAUSE AND NATURE. By EUGENE L. OPIE, M.D., Associate in Pathology, Johns Hopkins University; Fellow of the Rockefeller Institute of Medical Research. Philadelphia and London: J. B. Lippincott Co., 1903.

THE author has devoted this book almost entirely to the study of the conditions peculiar to the pancreas, and has omitted the forms of disease common to many other organs. Thus he avoids descriptions of acute suppurative lesions, tuberculosis, malignant growths, etc., but carefully considers hemorrhagic and gangrenous pancreatitis, hyaline degeneration with and without involvement of the islands of Langerhans, fat-necrosis, the relation of pathological change in the pancreas to diabetes, hæmochromatosis, diabète bronzé. The author goes at length into the anatomy, both gross and histological, and discusses the various anomalies of the pancreas, showing the relation that the anatomy and even the anomalies may have to disease, especially to the acute forms of pancreatitis.

Acute pancreatitis is divided into two forms, acute hemorrhagic and gangrenous, but the author very clearly points out that these forms are merely different degrees of the same process, with the same etiological factor at work. Of special interest is the ingenious theory in regard to the production of acute pancreatitis by the injection of bile into the greater pancreatic duct.

The mechanism of this process is explained and illustrated by a diagram. The author considers acute pancreatitis as secondary to

biliary stone. The gallstone is passed through the common duct, arrives at the duodenal opening, and is there arrested temporarily or permanently. In those cases where the stone is of such size that it blocks the duodenal opening without blocking either the common biliary duct or the pancreatic duct, it is possible for bile to flow or even be forced directly into the pancreatic duct with resulting hemorrhagic or gangrenous pancreatitis. So many correlating conditions must be fulfilled to produce this result that the theory amply accounts for the rarity of the disease.

Of the chronic forms of inflammation of the pancreas, two are recognized, namely, the interlobular and interacinar, while the author gives a separate chapter to a chronic hyaline degenerative process confined to the islands of Langerhans, resulting in the production of an unknown substance, and associated with diabetes in a very large percentage of cases. The weight of argument backed by post-mortem findings tends to prove that whatever impairs the health of the islands of Langerhans produces diabetes mellitus; that the interlobular form is to a less extent a factor, as there is in this type of inflammation small tendency until late in the disease to involve the interacinar islands. The interacinar form ranks next as a causative power, while hyaline degeneration primarily attacking the islands has the highest percentage of associated diabetes. The author recognizes that in more than half of all cases of diabetes the cause lies in a destructive lesion of the pancreas.

The volume shows evidence of much work carefully done, and the author's conclusions are arrived at by solid reasoning from experiments and symptoms. J. N. H.

A Nurses' Handbook of Obstetrics, for Use in Training Schools. By Joseph Brown Cooke, M.D. Philadelphia: J. B. Lippincott Co., 1903.

This is without question one of the most satisfactory books for nurses yet published. As the author remarks in his preface, there is no dearth of small volumes upon this subject, but most of the writers have attempted to speak in such a way that they might appeal to the patient herself even more than to the nurse. On the other hand, the present volume keeps its intent, as expressed in its title, consistently in remembrance, and addresses a trained audience exclusively. It is most clear, yet concise, and though at first sight there may be a feeling that the author has gone a little too deeply into the subject, we feel sure that consideration will compel agreement with his belief, that the more intelligent the nurse is theoretically, the greater aid she will be to the physician and the safer guardian to the patient.

An underlying aim, and one with which we have the most hearty agreement, is noticeable throughout the book, namely, to prepare the nurse to cope with the complications which may arise in the absence of the physician. Let it not be understood that any encouragement is offered to influence the nurse to assume undue responsibility, since in no instance is this true, the intent merely being to furnish intelligent "first aid" while awaiting the doctor's arrival. The book is well illustrated, and this fact, together with the appended glossary, enhances its value.

One of the noteworthy features is the chapter upon the preparation of the patient for labor, together with its armamentarium as well as the directions for the preparations of instruments, dressings, etc., for major obstetrical operations.

Throughout the book the necessity for asepsis is emphasized, though the value of the rubber glove is not dwelt upon.

We can most heartily recommend the book to the attention of all superintendents of training schools. W. R. N.

THE PRACTICAL MEDICINE SERIES OF YEAR BOOKS. Edited by GUSTAVUS P. HEAD, M.D., Professor of Laryngology and Rhinology, Chicago Post-Graduate Medical School. Vol. VIII., pp. 326. Chicago: The Year Book Publishers, 1903.

THIS volume has for its subjects Materia Medica and Therapeutics, Preventive Medicine, Climatology, Suggestive Therapeutics, and Forensic Medicine; and for authors George F. Butler, Henry B. Favill, Norman Bridge, Daniel R. Brower, and Harold N. Moyer. Over other Year Books this series presents the advantage of small size and more frequent appearance, containing about the same material, the value of all consisting in careful selection, accurate perspective, and judicial comments. For convenience of reference it would be well if the year could be gathered into one volume with an accurate and complete index; for reading perhaps the present is more advantageous. The collaborators have attacked their task with commendable thoroughness, and, while the careful reader will find but little with which he is familiar, perhaps repetition will fix it more indelibly upon his memory. The sub-editor's judgment may not always accord with that of the reviewer as to the importance of work, and he might even suggest that it be brought down to a later date. However, the results are good, and as a review it may take its place with others and find its sphere of usefulness. As we recall some of the earlier volumes, the press-work and paper have improved to the point where, like the contents, they are satisfactory. R. W. W.

A MANUAL OF OBSTETRICS. By A. F. A. KING, A.M., M.D., Professor of Obstetrics and Diseases of Women and Children in the Medical Department of the Columbian University, Washington, D. C., and in the University of Vermont, etc.; Ninth Edition. New York and Philadelphia: Lea Brothers & Co., 1903.

A REVIEW of this well-known work seems hardly to be required. Few, if any, students are unacquainted with the work, and its popularity is evidenced by the appearance of this, the ninth edition since 1882. Its size has been gradually enlarged until at present it comprises a volume of over six hundred pages, an increase of two hundred pages since the fourth edition was published. The advance made by medical science during the past twenty years has compelled the introduction of considerable new material as well as the practical rewriting of the chapter on Puerperal Septicæmia. We can, from our own personal experience as a student, most heartily recommend the work. W. R. N.

INTERNATIONAL CLINICS. Edited by A. O. J. KELLY, A.M., M.D., of Philadelphia, Pa., U. S. A., with the aid of Collaborators and Correspondents. Vol. II., pp. 311. Thirteenth Series. Philadelphia: J. B. Lippincott Co., 1903.

THE present volume opens with an excellent symposium on the "Summer Diarrhœas of Children," in which the subject is presented from the viewpoints of bacteriology, pathology, symptomatology, and therapeutics. It is well balanced, temperate in statement, instructive, and comprehensive, well worthy of the names it bears: Conn, Hand (Jr.), Cotton, Westcott, Nicoll (Jr.), and Marfan. Disease of the Pancreas, of which the medical aspect is taken up by Opie and the surgical by Deaver and Müller, is creditable to the authors. Here we find well-known opinions reiterated, but in condensed form, and yet from both sides the facts as presented are easily grasped and the importance of a comparatively new subject demands its acquisition. Treatment is introduced by Levi on "Trunecek's Serum in Arteriosclerosis." Although his claims are perhaps too much tinged with enthusiasm, yet the insidious character of the condition, its widespread and unfortunate results when fully developed, and its baneful effects as a complication urge the fullest investigation of any method which may promise anything of arrest or mitigation. Gaston's short "Practical Notes on the Prophylactic and Curative Treatment of Influenza, Malaria, Erysipelas, and Ozæna" are correctly entitled. Bouchard pleads for local, when possible, in contradistinction to general medication, and cites numerous instances in which normal tissues may be spared

inundations of active medicaments. His list might be advantageously further extended. Taylor recapitulates the indications for and conduct of the rest treatment. Even if verbose, this is a good presentation of the subject, and for those to whom the master's writings are not familiar its study is to be commended. In the "Etiology, Prevention, and Treatment of a Common Cold" Haig adjusts his theories to a practical end, although certain difficulties in the way of full acceptance suggest themselves. The remainder of Medicine is devoted to the heart, and Bishop, Poynton, and the always instructive Satterthwaite present excellent papers. Surgery covers Hemorrhoids (Gay), Abdominopelvic Diagnosis (Bishop), and Traumatic Epilepsy (Roncali), each adequately presented, although containing nothing of novelty. Hamilton traverses the question of "Cirrhosis of the Liver in Children," insisting upon the rôle of certain infectious diseases as causative agents. With his opinions we are in accord, and would suggest a reading of the remarkable work of Boix in this connection, which demonstrates the influence of other agents, some of which may come into the life-history of the child. In Obstetrics and Gynecology Goffe on "Sterility," Lockyer on "Ectopic Gestation," and Palmer on "Cervical Lacerations" are particularly satisfactory. The single paper on Ophthalmology is by Landolt on "Surgical Intervention in Paralysis of the Ocular Muscles."

We have noted the articles in this volume *in extenso* because all possess so great merit that omission would be unjust to author and editor. The standard, high as it was, set by the new editor is fully maintained, and we trust that others will find the same pleasure and profit in its reading which we have experienced and gained.

R. W. W.

THE MEDICAL EPITOME SERIES. MEDICAL JURISPRUDENCE: A MANUAL FOR STUDENTS AND PRACTITIONERS. By EDWIN WALLER DWIGHT, M.D., Instructor in Legal Medicine, Harvard University. Series edited by V. C. PEDERSEN, A.M., M.D. Philadelphia and New York: Lea Brothers & Co.

THE difficulties of preparing a brief manual which rises above the level of a quiz compend, yet answers the same purpose, and in which the essential facts concerning the subject are stated shortly and intelligently, are manifest; but notwithstanding these difficulties the present number of the Epitome Series fulfils the required conditions and fully realizes the editor's hopes. The text is continuous, but so paragraphed that any division of a subject may be readily referred to; while at the end of each chapter is placed a series of questions, thus rendering the book suitable for quizzing. The

volume opens with a short chapter on the general principles of medical jurisprudence, after which follow chapters on special divisions of the subject. Whenever it has been possible, tables have been inserted. Although many tables and much space have been devoted to the distinguishing features between human and animal blood, no mention is made of the newer methods for diagnosing human blood by the action of the anti-bodies. W. T. L.

THE MEDICAL EPITOME SERIES. MICROSCOPY AND BACTERIOLOGY: A MANUAL FOR STUDENTS AND PRACTITIONERS. By P. E. ARCHINARD, A.M., M.D., Demonstrator of Microscopy and Bacteriology, Tulane University of Louisiana, Medical Department. Series Edited by V. C. PEDERSEN, A.M., M.D. Philadelphia and New York: Lea Brothers & Co.

IN a book of this size and scope it is scarcely possible and indeed hardly desirable to do more than state the essential facts and accepted doctrines concerning the subject. The present work, therefore, represents in a way an abstract of the larger and standard text-books upon bacteriology and furnishes the outlines for a more extended and precise study of the subject. The first part of the book is devoted to a short review of the fundamental principles of bacteriology and to a description of the more common technical procedures, under which are included the preparation of media, sterilization, and a few staining methods; the later portions, on the other hand, deal with the morphological and physiological characteristics of most of the important pathogenic bacteria. The B. dysenteriæ receives due recognition. At the end of each chapter is appended a series of questions, which adapts the book for quizzing. There are 74 illustrations, among which are a few borrowed from Abbott's excellent plates. W. T. L.

A MANUAL OF BACTERIOLOGY. FOR STUDENTS AND PHYSICIANS. By FRED. C. ZAPFE, M.D., Professor of Histology in the College of Physicians and Surgeons, and Professor of Pathology, Bacteriology, and Hygiene in the Illinois Medical College, Chicago. Lea's Series of Pocket Text-books, edited by BERN. B. GALLAUDET, M.D.

THE selective faculty necessary in the preparation of as condensed a manual as the members of this series necessarily must be has been on the whole judiciously exercised in the present instance. Methods of sterilization and its surgical importance, the preparation

of culture media, staining and culture methods, and the technique of animal inoculation and post-mortem examination are treated with sufficient detail; and the description of bacteria, particularly those which are clinically most important, leaves nothing to be desired, while the plates and engravings are excellent and well chosen. Theoretical questions, on the other hand, are put off in a most cursory fashion, the section devoted to such a subject as immunity, for example, being little more than an expanded glossary of technical terms, or at best a suggestive list of subjects for the student's guidance. In the discussion of antitoxins and other bacteriological products employed for therapeutic purposes the author strikes a conservative note; diphtheria is still, in spite of much good experimental work that has been done in this line, the only disease in which positive results can be expected from the use of antitoxin.

The text contains certain discrepancies that cannot fail to perplex the student and seem to call for some explanation. Thus on page 274 we read that "thus far it has been impossible to produce typhoid fever experimentally in animals," and later, on page 282, under immunization, that "it is possible to immunize animals against typhoid." Again, the assertion on page 256 that "cholera is pathogenic for men only" does not harmonize with the statement that "the serum of the blood of animals that have recovered from cholera contains a substance which has decided bactericidal properties." We assume that in each case the identical disease is meant, and if so, the statements either require revision or they are to be taken in a relative sense only. Perhaps the looseness of statement may be partly accounted for by a similar laxity in the use of words revealed, for example, in the case of "tubercular" and "tuberculous," which occur synonymously—sometimes in the same sentence —yet the distinction between the two is clear and, one would think, ought to appeal with special force to a pathologist. "Postmortemed" may be convenient, but is scarcely musical, and is certainly an avoidable barbarism that ought to be relegated to the laboratory, where it was presumably evolved. R. M. G.

AMBULANCE WORK AND NURSING. A Handbook on First Aid to the Injured, with a Section on Nursing, etc. Chicago: W. T. Keener & Co.

No pains have been spared to make this work attractive, as it is handsomely bound in cloth and gilt, and there are many illustrations, for the most part from photographs. Particularly good are those showing the different methods of bandaging. It is written for lay readers, and for such should prove of interest and value, though

for the professional man there is little to be gleaned from its pages. The book consists of some chapters on anatomy for beginners, sections on first aid in all emergency cases, bandaging and rules for transporting patients, including the litter drill as used in England. The work is evidently intended for English readers, in fact, and the section on nursing is in many respects not applicable to conditions existing in America. G. M. C.

ADENO-MYOME DES UTERUS: AUS DEM PATHOLOGISCH-ANATOM. ARBEITEN. Herrn. Geh. med. Rath DR. JOHANNES ORT zu Feier seines 25 jährigen Professoren-jubiläums gewidmet von den Göttinger Assistenten, Schillern und Freunden. Von THOMAS S. CULLEN, M.D., Hülfsprofessor der Gynäkologie au der Johns Hopkins Universität.

IN a carefully worked out and excellently illustrated monograph Cullen discusses the pathological histology, etiology, and symptomatology of adenomyoma of the uterus. The work is based on an exhaustive and accurate study of 19 cases of this affection which occurred at the Johns Hopkins Hospital among 700 instances of myomata of the uterus.

The class of tumors under discussion is divided into three groups:

I. Adenomyomata with preservation approximately of the normal shape of the uterus.

II. Subperitoneal or interligamentary adenomyomata.

III. Submucous adenomyomata.

The tumors in general consist of a growth of smooth muscle fibres arranged in bundles, between which are scattered glands and ducts embedded in a stroma exactly like that of the normal uterine mucosa. The glands are also identical with the glands of the mucous membrane lining the uterine canal. In the first class of cases, where the growth extends diffusely through the uterine wall, the muscle bundles of the tumor fuse imperceptibly with the muscle bundles of the wall of the uterus, and a direct connection is often to be made out between the glands embedded in the growth and the glands of the uterine mucosa. Frequently several glands or a single gland can be traced as it makes its way down from the mucous membrane between the muscle bundles into the adenomyomatous growth; and in such cases a small amount of the normal stroma of the mucosa surrounds the gland and isolates it from the muscular tissue.

Occasionally small cystic spaces are seen which are lined by what appears as a mucous membrane indistinguishable from the normal uterine mucosa, while in the subperitoneal tumors very large cysts may occur, the cavities of which are filled, as a rule, with bloody or chocolate-colored fluid. The origin of the bloody fluid is explainable

on the supposition that the mucous membrane lining the cysts undergoes much the same alterations during menstruation as the mucous membrane lining the uterine cavity, and at these periods blood escapes into the cyst. The tumors may be found in the wall of the cervix as well as in the wall of the uterus.

The origin of the glandular portion of the growth can be directly traced to the mucous membrane of the uterus, and whether the tumors are submucous, subperitoneal, or ligamentary in position, the glandular portion arises from some part of the Müllerian duct. The older theory, advanced by von Recklinghausen, that the remains of the Wolffian body furnish tissue from which the glands may spring, is no longer tenable, and the author's view is upheld both from a study of his own cases and of those reported recently by others. The etiological factor concerned in the production of the tumors is uncertain, but it would at least seem that the myomatous portion of the growth develops first, just as adenocarcinomata may arise from normal uterine mucosa, so they may take their origin from the glandular islands or cysts of the adenomyomata; but the tumors themselves are essentially benign, as can be demonstrated both from a histological standpoint and from clinical observations. The symptomatology is dependent in large part upon the size and position of the tumors. Menorrhagia and pain in the back are frequently noted. Other symptoms may arise from pressure produced by the tumors. The condition was found oftenest in women between the thirtieth and sixtieth years of life. The prognosis is good even after partial myomectomy. W. T. L.

"Golden Rules" Series. For Diseases of Children. By George Carpenter, M.D. Of Hygiene. By F. J. Waldo, M.D. Of Aural and Nasal Practice. By P. R. W. de Santi, F.R.C.S. Of Refraction. By Ernest E. Maddox, M.D. Bristol: John Wright & Co.

This series of little books, none of them exceeding a hundred pages in length, is an attempt to present in the most concise form some of the more important principles of the different branches of medicine and surgery whereof they treat. Their authors are all men of experience and standing in the profession, and they have succeeded in condensing in these little volumes what may be described (as the title of the series would indicate) as the absolutely essential rules of practice in the different specialties. It may be truthfully said of them that while much is left out that might with value have been put in, there is nothing inserted which should have been left out. They are, therefore, of much more value to the student than is the general run of such vest-pocket companions. F. R. P.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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On the Production of Specific Cytolytic Sera for Thyroid and Parathyroid, with Observations on the Physiology and Pathology of the Parathyroid Gland, especially in its Relation to Exophthalmic Goitre.—In 1902 GONTSCHARUKOV claimed that he had been able to produce a specific antithyroid toxin by the injection, at intervals of four weeks, of an emulsion of dog's thyroids into the subcutaneous tissue of a sheep. By the injection of the blood serum of this sheep, which died during the experiment, into the circulation of other dogs certain tetanic symptoms resulted which he thought indicated the existence of a specific antithyroid toxin.

W. G. MACCALLUM (*Medical News*, October 31, 1903, p. 820) conducted a series of experiments to control these results. He points out first that the destruction of the thyroid alone produces disturbances of metabolism, which appear slowly and are characterized by the symptoms known as myxœdema. Destruction of the parathyroid alone, on the other hand, produces the acute rapidly fatal nervous phenomena which have so long been thought to be due to the extirpation of the thyroid. These phenomena are tetany and polypnœa. Until recent years the early fatal results following thyroidectomy had been attributed to the extirpation of the thyroid, while in reality they were due to removal of the parathyroid glands, which are small and inconspicuous and usually embedded in the tissue of the thyroid.

In his investigation MacCallum made repeated injections of an emulsion of dog's parathyroids into the peritoneal cavities of geese. At intervals the centrifugalized serum from the defibrinated blood of these geese was injected into the peritoneal cavity of dogs. The symptoms produced in these dogs were not at all striking. Emaciation and a

cachexia developed in a number of them. These symptoms, however, occurred also in dogs injected with normal geese serum. In one case convulsive twitching of the muscles resulted without dyspnœa. After the injections a large number of the dogs were killed at varying intervals and their thyroids examined histologically. In none of the cases could a microscopic change be made out. The writer therefore concludes that the attempt to produce a cytotoxin capable of destroying the cells of the thyroid glands *in situ* has not been successful. He believes, however, that a material was probably produced in the blood of the geese capable of combining with the normal secretion of the dog's thyroid or parathyroid and neutralizing it, thus allowing the symptoms which ordinarily follow extirpation of these glands to occasionally appear in a mild degree. MacCallum thinks that his experiments throw considerable doubt on Gontscharukov's results, at least on the interpretation of them, for one would not expect tetanic contractions as an effect of the destruction of the thyroid by a specific serum.

The writer was led by the above work to carry on some investigations with the view of determining the rôle of the parathyroid glands under normal conditions and in disease. Dogs were used in the experiments. The parathyroids were removed in a number of animals. The symptoms of the parathyroidectomized animals were briefly as follows: After symptoms of unrest, with slight twitchings of the muscles, the animal rapidly develops violent tetanic spasms. It experiences difficulty in walking and periodically falls to the floor in an epileptiform convulsion. There is marked trismus, and the eyes appear to project owing to retraction of the upper lid. The respirations are greatly increased, even up to 200 to 250 per minute, but there is no cyanosis. There was no specially increased heart action. The animal may die at the height of the attack, or the convulsions may gradually become less violent, death resulting from exhaustion.

Three problems present themselves: (1) Are these symptoms due to absence of some necessary secretion of the parathyroid? (2) Are they due to some poison produced somewhere in the body and circulating in the blood, but which, in the normal animal, would be neutralized by the parathyroid cells themselves or by their secretions? (3) Is this poison absorbed from the intestine; is it the result of metabolism of certain groups of cells, or is it the product of muscle or nerve metabolism? As a result of some ingenious experiments the writer concludes that while it seems improbable that the parathyroids secrete a necessary material which is circulated in the blood, the lack of which produces the disturbances described, it does seem probable that they produce a material which neutralizes poisons produced elsewhere, poisons which if not so neutralized may be mechanically washed out with the relief of the symptoms.

The parathyroids were shown to have no influence on nitrogen metabolism, whereas it was demonstrated that the nitrogen output was markedly affected by removal of the thyroids.

There is no very convincing evidence advanced by the writer to show that the parathyroids are in any very close way associated with the etiology of exophthalmic goitre. Parathyroidectomy is followed by muscle spasms, and muscle tremor is a symptom of exophthalmic goitre. Occasionally there is slight prominence of the eyes following the operation. Further than this there is no particular resemblance. It is

worthy of note, however, that an examination of the parathyroids embedded in the excised thyroid glands of exophthalmic goitre patients all showed atrophy, and in two instances the glands showed very distinct degenerative changes in the parenchyma cells with overgrowth of connective tissue.

The Thick-film Process for the Detection of Organisms in the Blood.—Ronald Ross (*Thompson Yates and Johnston Laboratories Report*, 1903, vol. v., part I., p. 117) uses a very large drop of blood in this method, and does not attempt to spread the blood out in a thin layer on the cover-glass. No attempt at fixing the hæmoglobin is made. The thick film of blood on the slide is merely dried, and then very gently washed with water until all color has disappeared. For staining purposes only aqueous solutions of the stains are used. Ross now uses the following stock formulæ for staining: (1) Eosin, 1 gram; water, 1000 c.c. (2) Medicinal methylene blue, 10 grams; sodium carbonate, 5 grams; water, 1000 c.c. After washing out the hæmoglobin as described above, a drop or two of the eosine solution is now placed upon the cover-glass without previous drying, and allowed to remain for one minute. Then a drop or two of the methylene blue solution is run on and allowed to remain for fifteen to thirty seconds. Finally the coverslip is washed very gently with a stream of water for a minute or two, dried and mounted for examination.

Ross uses this method for detecting the presence of malarial organisms where they are very few, and also for trypanosoma. In one case of malaria where only one parasite could be found in a field by the old method, by the new one as many as eighty could be found. The malarial parasites stain blue and the nuclear chromatin a distinct red. The red cells are unstained, owing to their hæmoglobin being washed out. Bell and Laing advocate the use of this method for the detection of plague bacilli in the blood.

The Tendo-Achilles Jerk and Other Reflexes in Diabetes Mellitus — Williamson (*Review of Neurology and Psychology*, October, 1903, p. 667) states that in diabetic mellitus, as in locomotor ataxia, the tendo-Achilles jerks may disappear before the knee-jerks are lost. His results in the examination of these reflexes in 50 diabetics was as follows: (1) In 19 both Achilles-jerks were absent. In these cases both knee-jerks were present in 8; one present and one absent in 3; both absent in 8. (2) In 2 cases one Achilles-jerk was absent and one present. In these cases both knee-jerks were present. (3) In 29 cases both Achilles-jerks were present. In this series both knee-jerks were present in 28, and one knee-jerk present and one absent in one.

The writer points out that the knee-jerks are much more likely to be absent in hospital than in private cases, in which the conditions of life are better. Thus, in the former he found both knee-jerks absent in 49 per cent. of the cases, while in the latter both were absent in only 12 per cent. He finds that the wrist-jerks are usually absent where the knee-jerks are absent. The causes for the disappearance of the deep reflexes are not discussed. The superficial reflexes—plantar, abdominal and epigastric—are present in practically all cases. It is stated that in the severest cases the abdominal and epigastric reflexes are usually much increased. The plantar reflex is of the normal type.

SURGERY.

UNDER THE CHARGE OF

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The Diagnosis of Bone and Joint Tuberculosis.—LUDLOFF (*Centralblatt für Chirurgie*, September 5, 1903) states that in the treatment of bone and joint tuberculosis, the choice between resection and iodoform injections remains still but a matter of personal desire. The *x*-ray picture plays a large rôle, especially in cases of beginning tuberculosis, where one should try to recognize the trouble early enough to prevent its extension into the neighboring structures. The author reports in detail three cases in which an early diagnosis was made by means of the *x*-rays. Every case when seen early should be studied first in regard to its etiology, and the first question to be determined is, Was the synovial membrane infected secondarily from a lesion in the bone, or *vice versa;* or were both infected at the same time from the same place? It is to be remembered that the epiphysis is the point of entrance for the numerous nutrient bloodvessels into the bone, and that this point lies just on top of the synovial membrane. For a solution of these questions more accurate examination of the epiphysis and synovial membranes are necessary. It is still to be determined whether iodoform injections are of the same value on the bone as they are on the membrane near an enlargement of the bony parts of a diseased joint by reason of multiple deposits. If resection is not done, the only other method of any value whatever consists in iodoform injection.

A New Operation for the Relief of Wryneck.—ULLSTEIN (*Centralblatt für Chirurgie*, August 15, 1903) sums up his remarks by placing the greatest importance on the following questions, which he asks and answers as follows:

1. How much of the muscle should be shortened? Of course, this varies with each particular case, and must be graduated according to the previous differences in length, and, as a rule, it will vary from 4 cm. to 8 cm.

2. In which part of the muscle should shortening be made? The shortening should be made in the upper portion above the entrance of the nerve supply. As to the best method, simple resection of the muscle is not satisfactory, for the reason that if this be done the muscle lacks the ability to contract, and for that reason it is essential to do resection, which not only does not interfere with the continuity of the fibres, but also does not interfere with the nerve supply. A bandage should be retained in place for about twenty days after the operation, at the end of which time it may be safely removed, and, as a general rule, the results are satisfactory.

New Method of Treatment for Backward Dislocation of the Hip.—ELGART (*Centralblatt für Chirurgie*, August 22, 1903) states that the method consists in anæsthetizing the patient, who is then placed upon the floor, and then the operator should get down on one knee and place the dislocated limb over his thigh, and then, while an assistant securely holds the patient's pelvis, the operator should grasp the ankle with one hand and make traction, while with the other hand clasped on the thigh, the femur should be rotated inward and the leg pressed outward. In a bad case of backward dislocation this was followed on the first trial by perfect reposition. The method differs from Kocher in that, by placing the dislocated limb over the operator's thigh, much more pressure can be brought to bear, and, as a result, the reduction becomes easier.

The Lotheissen Method of Radical Cure for Femoral Hernia.—GILLI (*Centralblatt für Chirurgie*, August 8, 1903), after giving in detail the method employed, states that his experience has shown that the separation of the falciform process and of Poupart's ligament in these cases of irreducible hernia distinctly simplifies the condition of the hernial sac, as well as shortens the time of operation. The technique consists in making an incision parallel to Poupart's ligament, then exposing the sac of the hernia, which should be separated from its attachments at the edge of the ring. Then the aponeurosis of the external oblique should be separated and a Kocher sound should be passed between the sac and the falciform process until its tip appears at the abdominal side of the inner crural ring. The inguinal ligament and the falciform process can then be separated on the sound. After this separation the sac of the hernia may be easily isolated. The advantages of the method are: 1. That the separation is accomplished under the eyes of the operator, and there is no necessity for blind, uncertain work with a hernia knife, and any bleeding which may occur can be easily controlled. 2. In case of bad strangulation, the contents of the hernia are exposed to full view and can be thoroughly inspected before being replaced within the abdominal cavity. Experience has shown in cases where general anæsthetics are contraindicated that this method may be used with great satisfaction under local anæsthesia.

THERAPEUTICS.

UNDER THE CHARGE OF

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Physostigmine in the Treatment of Intestinal Atony.—GUGLIELMO CURLO concludes from his laboratory and clinical experiments that this substance (1) manifests an excitant action upon the intestinal

muscles which results in spasm with increased peristalsis, (2) it is especially indicated in all forms of coprostasis due to intestinal atony, being contraindicated in spastic conditions, acute and chronic intestinal catarrh, and mucomembranous enterocolitis. (3) On account of its spastic action in increasing the tone of the intestinal muscles it is an excellent remedy for meteorism. (4) The maximum daily dose which can be given without symptoms of intolerance is from $\frac{1}{10}$ to $\frac{1}{16}$ grain, but the usual dose is one-half of this quantity. (5) Myosis and salivation mark the point of tolerance of the remedy. (6) The preferable salt is the salicylate, and it is best administered as a pill.—*La Riforma Medica*, 1903, No. 37, p. 1009.

Action of Hæmolytic Sera —Dr. Robert Muir discusses this question from several points of view, detailing a number of experiments and methods of experimentation from which he derives the following conclusions: (1) Immune body and the complement differ markedly from each other in their mode of combination and in the firmness of their union. (2) Immune body can be in part separated before hæmolysis from red corpuscles containing multiple doses. (3) When red corpuscles containing multiple doses of immune-body are hæmolyzed by the minimum dose of complement the surplus molecules of immune-body remain attached to the receptors of the red corpuscles and can be in part separated. (4) Complement either after its union directly with cells or indirectly through the medium of an immune-body cannot be separated again. (5) Each dose of immune-body taken up by red corpuscles, in addition to the hæmolytic dose, leads to the taking up of an additional amount and corresponding amount of complement necessary for hæmolysis may be used up. (6) When red corpuscles containing multiple doses of immune-body are fully saturated with complement, immune-body can still be separated. The immune-body thus obtained comes off alone and not in the combination I. B. + C.—*Lancet*, 1903, No. 4172, p. 451.

Nauheim Treatment in Chronic Affections of the Heart.—Dr. Leslie Thorne, basing his conclusions on an experience with the Nauheim methods during the past seven years in London, divides his cases of heart disease into four groups: (1) those who will be cured; (or) benefited very greatly by the treatment; (2) those which cannot be cured, but can be greatly benefited; (3) the doubtful cases, and (4) unsuitable cases. Of the cases which will be cured or benefited very greatly by the treatment, the dilated, enfeebled, and irritable heart, a sequela of influenza, is one of the most promising, and it is also one that, in many instances, resists treatment by drugs, rest, or change of air, so that the unfortunate sufferer often becomes a chronic invalid with nothing but a broken and almost useless life to look forward to. He believes it no exaggeration to say that the Nauheim treatment often gives a new lease of life to these cases, though if they are of a severe type they may require two, or even three courses, at intervals of nine months to a year to restore them to health. Another class of cases which belong to the first group is that of the dilated and enfeebled heart produced by the raised arterial tension present in the circulation of patients suffering from rheumatic or gouty diatheses. This slowly but continuously acting pressure produces in time an overloaded and overworked heart,

and thereby an increasingly impure blood supply and a progressive weakening of the cardiac systole. It is almost impossible to cure these cases absolutely and permanently; the very fact that the poison is manufactured in the system and can be eliminated only by a careful diet and well-regulated life often leads to a recurrence of the heart symptoms in time and makes it almost a necessity that the patient should undergo a course of treatment regularly every twelve months for two or three years, and then perhaps every second or third year. He says there is no greater mistake than to lead these people to believe that one course will cure them completely, though young patients suffering from mild forms of rheumatic and gouty hearts, without valvular disease, will often remain in good health for several years after a single course, especially if a careful dietary and habit of life are followed. Cases of cardiac enfeeblement from excessive smoking and prolonged illness, such as typhoid fever and malaria, belong also to this group, the Nauheim treatment being in these cases a most valuable aid to such methods of cure as rest, tonics and change of air, and producing a much more rapid return to health than could otherwise be expected. (2) In this group he ranks those cases of both rheumatic and gouty origin in which the valves have been permanently injured, and signs of commencing cardiac failure, such as headache, shortness of breath, palpitation, cyanosis, and pain, are present. It is self-evident that patients of this class cannot be cured and that one course of baths will not produce a permanent improvement, but it will undoubtedly produce a much more lasting and satisfactory one than any other treatment. (3) Authorities differ greatly as to the nature of the cases that should be included in this group. The author's experience of the treatment has led him to believe that unsuitable patients are those who are, and have been, habitually heavy drinkers, those whom one believes to be suffering from syphilitic affection of the heart, those suffering from marked degeneration of the vessel walls, those exhibiting typical symptoms of aortic regurgitation, and very old people. The chronic heart case usually met with in hospitals, broken down by a long struggle to work when unfit and accustomed to bad and even insufficient food, is also one of the most unsatisfactory for the Nauheim treatment.—*Lancet*, 1903, No. 4168, p. 153.

Sublamine in the Treatment of Parasitic Scalp Diseases.—Dr. William S. Gottheil, in reporting an extensive experience in ringworm in a large institution, finds that this remedy was more rapid than the bichloride because of its greater penetration and because it can be employed in stronger solutions, being less irritant. The solutions employed were 1:750 or 1:1000.—*The Medical News*, 1903, vol. liii. p. 735.

Sciatica; its Nature and Treatment.—Dr. Wm. Bruce contributes some revolutionary ideas to the etiology and pathology of sciatica, based on deductions of some 418 cases which have been under his observation during the past thirteen years. He believes that, after all, sciatica is a disease of the hip-joint and not a neuritis of the sciatic nerve, and that it is connected with the gouty or rheumatic diseases, and may be called a monoarticular rheumatic arthritis of the hip. The reasons for his deductions he presents, and his conclusions lead him to a modification

in the treatment. Rest of the joint is one of the most important features. Blisters may be of some service, and sedative lotions and fomentations are sometimes valuable. Massage and electricity are contraindicated. Antigout and antirheumatic remedies are recommended. Turpentine has proven of benefit when taken internally.—*Lancet*, 1903, No. 4173 p. 511.

The Treatment of Puerperal Sepsis.—Dr. Hiram N. Vineberg employs the ointment of colloidal silver (unguentum Credé) in cases when he can find no lesion which demands surgical intervention, believing that it is of some service either in aiding the system to eliminate the toxins produced or in some way counteracting their deleterious influence. It is certain that several desperate cases, in which this silver was employed by inunction, ended in recovery.—*Journal of Obstetrics*, 1903, No. 9, p. 325.

Dr. H. Fehling believes that this substance intravenously employed in sterile 2 per cent. solutions, 2 to 5 drachms for a dose, is of great value.—*Münchener medicinische Wochenschrift*, 1903, No. 33, S. 1409.

Treatment of Chorea by Ergot.—Dr. Eustace Smith calls attention to the use of ergot as a sedative in nervous diseases. He says in children particularly it acts as a very valuable sedative, believing this is an action due to its influence on the blood supply, on the nerve tissues in the spinal cord in part, or perhaps a direct sedative. No ill effects have ever been observed by him, although he gives doses of a drachm every hour for three weeks for children seven or eight years of age and doses of 20 drops or more for many months at a stretch. In chorea its action is not as reliable as Fowler's solution, but often arsenic is not well borne by many patients. Ergot acts more quickly than arsenic. He states a number of cases, saying that it is sometimes necessary to push the doses of the remedy, boys seemingly requiring larger doses than girls.—*British Medical Journal*, 1903, No. 2220, p. 133.

Aspirin in Chorea.—Dr. R. T. Williamson has recently been prescribing aspirin in thirty-five consecutive instances of chorea. Inasmuch as the mild forms of the disease have a tendency to recover without treatment in from six to ten weeks, his conclusions are given with a certain amount of reserve. In mild cases the aspirin has seemed to be of service, but other drugs have proven equally valuable. In severe cases he believes that the drug in from 10 to 15 grain doses, given three or four times a day, is of value. Aspirin cannot be considered as a specific of chorea, but it is well worth trying, particularly in severe cases. It is best given in powders in water to which a little lemon juice has been added.—*Lancet*, 1903, No. 4173, p. 526.

Antitoxin in Hay Fever.—Dr. Felix Semon gives his impressions on the efficiency of Dunbar's antitoxin in hay fever, some preliminary results of which he published in 1903. He bases his remarks on the observations of eight hay fever patients whom he has personally treated. These cases have been under his direct supervision and the patients themselves have not had charge of the treatment. Thus far he has not met with any untoward by-effects of any kind in his limited experience, and he thinks it reasonable that patients may be trusted with the self-

administration of the drug. In his trials he has used proportions of 1:500 antitoxin with equal quantities of normal horse serum. He has found that a single application will not ward off or cure the disease for any prolonged period and he says that he unfortunately cannot say that the remedy has in any sense acted as a cure in any cases.—*British Medical Journal*, 1903, No. 2220, p. 123.

Ichthyol in Pulmonary Disease.—Dr. Thomas Burnett has been using ichthyol extensively in pulmonary tuberculosis, in bronchitis, acute and chronic, and in pulmonary fibrosis. In 6 of the cases of tuberculosis he found that at the end of the first month of treatment the cough was less harassing, while in 5 others there was no improvement. At the end of the second month all of the patients, 13, showed distinct improvement with reference to cough, while in 8 expectoration was either less in amount, or both less in amount and easier in expulsion. By the end of the third month 7 of the patients were so far improved that no night-sweating was noted, and at the end of four months night-sweating was entirely absent in every case, while only a slight cough remained. Toward the end of the fifth month all of his cases showed a continued improvement, and at the end of the six months every one had gained in weight to the extent of from three to seven pounds, the average gain in weight being a pound a month. These 13 cases referred to were all of pulmonary tuberculosis. Ichthyol also relieved 2 of the patients with chronic bronchitis, and was very valuable in bronchiectasis. With reference to doses, the amount given was from 8 to 20 grains, four times a day, in capsule form, each capsule containing 4 grains of ichthyol. As a rule, the initial dose consisted of two capsules given four times a day, which was gradually increased until five capsules were taken four times daily. After a short time the patients became habituated to these large doses.—*Lancet*, 1903, No. 4171, p. 384.

Intravenous Injections of Iodoform in Advanced Phthisis.—Dr. T. W. Dewar presents a short preliminary report on the treatment of patients with advanced pulmonary tuberculosis by intravenous injections of an ethereal solution of iodoform. Apart from certain difficulties in the mode of injection, he has been able to throw from 5 to 45 minims of an ethereal solution of iodoform into the veins. Each injection should be prepared from fresh solutions. Frequently, following the use of a large dose, 20 to 25 minims, momentary unconsciousness may result. The method is still under experimentation, and the details are promised at a subsequent period.—*British Medical Journal*, 1903, No. 2238, p. 1328.

Radioactive Substances.—Dr. William Rollins says that for the present, when using radioactive substances in internal therapeutics, the substances themselves cannot be employed on account of their high price. Advantage can be taken, however, of radium salts, which, when dissolved in water, give off emanations. These can be collected in air in a gas-holder over mercury and used in internal medicine. When radium is dissolved 75 per cent. of its radioactivity is at once liberated, but 25 per cent. remains, consisting, for the most part, of A particles. When the first rush of liberated active substances is over, radium in

solution constantly gives off more emanations, which can be used in therapeutics. In using radioactive substances in disease of or near the skin, the depth of the diseased tissue to be affected must determine the distance at which the radiosubstance is placed upon the surface of the patient. The correct manner for treating a skin disease with these substances is to consider to what depth it is desirable to confine the activity. If to a slight depth, the radium should be almost in contact with the skin; the duration of the application always being shorter than when at a greater distance.—*Boston Medical and Surgical Journal*, 1903, vol. cxlix. p. 542.

Strychnine in Degenerative Diseases of the Nervous System.—Dr. G. M. Hammond discusses the action of massive doses of strychnine in the treatment of tabes, optic nerve atrophy, progressive muscular atrophy, and pseudomuscular hypertrophy. In tabes he reports four cases in which, under doses of from 1 to 3 grains daily, gradually administered, improvement in gait was readily marked. Of optic nerve atrophy, three cases are reported, in which doses of from $\frac{1}{2}$ to $\frac{3}{4}$ of a grain of strychnine a day arrested the degenerative process. In three cases of progressive muscular atrophy, one improved, one was held stationary, and the third was unmodified. In one case of pseudomuscular hypertrophy, a decided improvement was noted. His practice is to begin treatment with a moderate dose, usually $\frac{1}{40}$ to $\frac{1}{30}$ of a grain, three times a day, and to increase the daily dose by about $\frac{1}{30}$ of a grain. If symptoms of toxæmia develop, the dose is diminished. Doses of $\frac{1}{4}$ to $\frac{2}{3}$ of a grain, three times a day, are well borne by this gradual development in dosage.—*Boston Medical and Surgical Journal*, 1903, vol. cxlix. p. 228.

PEDIATRICS.

UNDER THE CHARGE OF

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AND

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The Relation of the Thymus Gland to Marasmus.—At the recent meeting of the British Medical Association, John Ruhrah (*British Medical Journal*, August 29, 1903, p. 455) reported the conclusions of his study of the post-mortem findings in eighteen cases of marasmus. He states that the most characteristic lesion of marasmus, except the wasting of fat and muscles, is not mentioned in any of the text-books on diseases of children—this is a wasting of the thymus gland. There are two conditions in which there is atrophy in infants—the primary cases, in which the cause is as yet unknown, and those that follow definite pathological conditions, or the secondary cases. The dividing

line cannot at this time be definitely drawn. For the present, he thinks, the cases should be divided on a pathological basis into those where there are lesions of definite diseases and those where there are no special and constant lesions, except the wasting of the muscles and the body fat, and, as he now points out, of the thymus gland as well.

After referring to the various theories that have been advanced by Baginsky, Bendix, Heubner and Rubner, Czerny, and French writers, he thinks we may safely assert that the trouble is not due to the amount of food ingested nor to the amount absorbed from the intestinal tract, but that it is due to the malassimilation of the food material in the body.

Whether the thymus gland has anything to do with assimilation or not, he is not prepared to say, but it is certain that the thymus bears a direct relation to the state of the nutrition of the body, as pointed out by Herard, Friedleben, and more recently by Mettenheimer. It increases in size until about the second year, when it remains stationary, growing relatively smaller, when compared with the size of the growing body. At puberty, when the body growth is largely over, it begins to atrophy, and at about twenty-five years of age, when the body has attained its full growth, it is gradually replaced by fatty and connective tissue.

The thymus gland weighs about twelve grams at birth. Histologically, it consists of a connective-tissue framework holding up a parenchyma consisting of a cortex and medulla, which are similar in structure, but the cortex contains normally many more cells than the medulla. The parenchyma consists of a network of endothelial cells resembling the reticular spaces of the lymphatic structures. This space is filled with lymphoid cells and also with a few neutrophilic and eosinophilic leukocytes, and with a few giant cells. Here and there in the structure are little islands of epithelial cells, with a peculiar concentric arrangement—the so-called Hassall bodies. They are the remains of the epithelium which forms the principal part of the thymus gland during embryonal development.

In his eighteen cases the average weight of the gland was twenty-two grams. Excepting the lesions of the terminal infection, the atrophy of the thymus was the only lesion found in any of the necropsies. In these specimens the fibrous capsule of the gland is thicker than normal, and the trabeculæ are also greatly thickened, the increased interlobular tissue frequently cutting the lobules into irregular masses. Sometimes there is more fibrous tissue than lymphoid structure, and in well-marked cases there is more reticular tissue than lymphocytes and leukocytes. In most of the cases there was an increase in size and a hyaline degeneration of the Hassall bodies.

A microscopic examination of the thymus gives a clear indication of the state of nutrition of the infant. In normal infants the structure of the thymus is unaltered; in moderate atrophy the cortex and medulla are not easily distinguished, while in severe atrophies the changes mentioned above are plainly seen. In secondary atrophy the change is one of degree, and the average weight of the gland was 3.41 grams. Administration of thymus gland in tablet form produced no appreciable changes.

In the discussion of this paper the question of "thymus death" was mentioned. EWART considered that the enlargement of the gland was not the only cause. Some other factor, such as pulmonary congestion

or gastrointestinal distention, of a more sudden nature, must be admitted. But the chief factor, in his estimation, was an individual cardiac weakness, which, in the adult, showed itself in the tendency to fainting and to sudden death. CARPENTER did not believe in thymic asthma or sudden death from an enlarged thymus. He believed death in these cases, in many instances, was in reality due to spasm of the larynx, which did not relax. Thymus death was often death from laryngismus stridulus. More attention should be paid to the nasopharynx, and thymic asthma would be a less fashionable explanation.

The Prophylactic Use of Diphtheria Antitoxin in School Children.—AUGUSTUS CAILLE (*Archives of Pediatrics*, October, 1903, p. 748) advocates an immunizing injection of diphtheria antitoxin for young children once or twice during the school year—for instance, in November and February—with the hope of preventing infection from primary diphtheria or croup; and, furthermore, with the hope of lessening the mortality of the severe forms of scarlatina and measles, a large percentage of such cases being complicated by diphtheria from the beginning or in the course of the disease.

It is well known that cases of scarlatina which show a complicating diphtheria from the onset are of a very grave type, showing an overwhelming sepsis, with delirium and circulatory failure. In measles, diphtheria is an early or a late complication, but the most serious form is diphtheritic croup. The mortality from scarlet fever or measles complicated by diphtheria is quite high, and the author's clinical experience suggests that this mortality can be markedly reduced by means of protective inoculations of diphtheria antitoxin.

During the past two years he has practically followed out this line of thought, and has immunized about twenty children twice during the school year. As far as figures can be of value, it can be stated that not one of these children contracted primary or secondary diphtheria, and in no case was there the least unpleasant or unfavorable reaction after the injection. The parents readily accepted the "protecting vaccination for diphtheria." Dr. Caillé has every confidence in the feasibility of this plan for communities in which diphtheria is endemic or epidemic.

Contribution to the Study of Scarlatinal Infection.—SZEKELY (*Jahrbuch für Kinderheilkunde*, 1903, Bd. vii., S. 789) reports a curious case of scarlatinal infection. In a family consisting of father, mother, and two sons, the elder boy, aged eleven years, developed scarlatina. The younger boy, three years old, was at once removed to the house of a friend, and was kept there nine weeks without having any communication, directly or indirectly, with his home. After the complete recovery of the older brother, following a rigorous disinfection of the house and its contents, the child was brought home. Ten days later this boy developed sore throat, fever, and a scarlatinal eruption which presented no peculiarity other than in beginning on the back of the right thigh. The disease pursued a normal course, was very benign, and ended in desquamation.

The interest in the case centred in the avenue of the infection, and a careful inquiry established the following facts: The older boy had presented during his illness some excoriations of the skin, which had been

treated with an ointment of oxide of zinc. It was discovered that some days after returning home the younger boy had developed a small eruption of vesicles on the right thigh, which had been smeared with the ointment which had been used for his brother, and which had not been destroyed at the time of the disinfection. Two days later the child had become ill with scarlatina, and, as already noted, the rash had begun upon the right thigh.

The author, therefore, asks if this case should not be considered as one of surgical scarlatina, the vesicles having served as a point of entry for the scarlatinal virus; and since the evolution of the disease was quite mild, he inclines to think that it proved to be, in effect, a sort of vaccination with a scarlatinal virus attenuated by its exposure to the mild antiseptic action of the zinc ointment.

Congenital Torticollis.—MAASS (*Zeitschrift f. Orthop. Chirurgie*, 1903, Bd. xi.) concludes from a study of forty cases of congenital torticollis, nearly all the subjects at the time of observation being under three years of age, that torticollis of intrauterine origin (due probably to lack of amniotic fluid) is rare; that the condition arises most frequently during birth, not as the result of tearing of the muscle, as Stromeyer contends, but from a traumatic necrosis of the muscle which has healed by formation of cicatricial tissue. This lesion, which is found principally in breech or forceps cases (80 per cent. in Maass' statistics), is due less to direct pressure upon the muscle than to its hyperextension.

When the distortion is too great to hope for regression, binder, massage and moist heat, Maass has recourse to Mikulicz's operation (partial resection of the muscle). With older children a post-operative orthopedic treatment should be continued for a long time, in order to correct secondary deformities of the vertebral column.

Polymyositis in Children.—SCHULLER (*Jahrbuch für Kinderheilkunde*, 1903, Bd. viii., S. 193) reports the observation of a child who, in the course of whooping-cough, was seized with fever, chills, headache, and dyspeptic symptoms. These phenomena, which lasted four days, during which the kinks were in abeyance, were replaced by swelling of the eyelids and a painful induration of the muscles of the face and neck. This rigidity, in turn, invaded the muscles of the thorax, back, abdomen, and, finally, those of the limbs. At the end of three weeks these painful contractures began to subside, and two months later the child was completely well.

In studying the features of this case, the author arrives, by exclusion, at a diagnosis of polymyositis, in favor of which he offers the following: The affected muscles were hard, infiltrated, both painful and tender, and were clearly outlined under the skin. The contractures persisted during sleep. Direct stimulation of the muscles by the faradic current produced slow contractions, while rapid contractions were produced by the indirect faradic or the galvanic current.

Five other cases of polymyositis in children have been found in the literature, and are abstracted in the paper.

Paralysis of Accommodation and of the Uvula following Mumps.—MANDONNET (*Annales d'oculistique*, 1903, No. 2) reports a case of this rare sequel of parotitis. The patient was a child aged nine years, who,

during convalescence from an attack of mumps, complained of dimness of vision, especially for near objects. Externally the eyes showed no abnormality except a slight dilatation of the pupil which could scarcely be called mydriasis. Both eyes, the right especially, were hypermetropic; vision was ½ in the right eye, ⅔ in the left, but correction of the hypermetropia of the right eye restored normal vision for distance. Near vision was very poor, the child being incapable of reading even the large characters, but was restored at once by the use of a + 4 D. spherical lens. The integrity of the eye-grounds being shown by the ophthalmoscope, the diagnosis of paralysis of accommodation following mumps was made. Paralysis of the uvula was also present, confirming the diagnosis.

Diphtheria Antitoxin in the Treatment of Aphthous Stomatitis.—Influenced by several suggestive experiences of Sangiovanni, Gaspardi, and Santi, DEL MONACO (*Revue Mensuelle des Maladies de l'Enfance*, August, 1903) was induced to use diphtheria antitoxin in the treatment of an infant one year old affected with a grave aphthous stomatitis which had produced a marked cachexia. The injection was promptly followed by a sensible amelioration in the general state, while at the same time the sublingual swelling, which had been unaffected by lotions of permanganate of potash and nitrate of silver, rapidly disappeared.

OBSTETRICS.

UNDER THE CHARGE OF

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Excessive Distention in the Wall of a Bicornate Uterus.—In the *Zentralblatt für Gynäkologie*, 1903, No. 45, JURINKA reports the case of a woman, aged thirty-two years, who complained of severe pain in the right lower portion of the abdomen. This pain increased greatly in severity.

On admission to the hospital the uterus was found to be anteflexed and rotated somewhat to the left, while upon the right side, and posteriorly, a tumor as large as an orange was present. This tumor was exceedingly painful on palpation. The left tube and ovary were normal, and the right tube and ovary could be distinctly palpated. Interstitial pregnancy was diagnosed, and the patient's intense and constant pain and the danger of uterine rupture made operation necessary. Accordingly, the abdomen was opened in the median line; the uterus, the tumor, and right tube and ovary were removed with the left tube, and a strand of iodoform gauze was carried through the stump of cervix for drainage. The patient made an uninterrupted recovery from the operation.

On examination pregnancy in the right cornu of a bicornate uterus was present. A fourteen weeks' ovum had developed, and a placenta was attached to the uterine wall. Especially noteworthy was the difference in the thickness of the uterine wall upon the two sides. Upon the right side the wall of the womb was 20 mm. in thickness, while on the left side, where the pregnancy existed, the wall of the uterus was but 3 mm. to 5 mm. in thickness. On microscopic examination the muscular tissue of the right side of the womb was much diminished in quantity and development, although the muscle cells and bloodvessels showed no especial irregularities. There was no hemorrhage.

A Case of Puerperal Tetanus.—WURDOCK (*Prager medicinische Wochenschrift*, 1903, Nos. 9 and 10) describes a case of tetanus in a woman, aged forty-two years, a multipara, who was delivered in Rubeska's clinic of a macerated and decomposed fœtus. Three hours afterward the placenta was manually removed and the uterus washed out. Fever developed for four days, but gradually disappeared, and the patient was discharged on the tenth day. On the next morning she could not open her mouth, had pain in the back and cramps in the back and neck, and these symptoms rapidly increased. Tetanus antitoxin was at once injected and urethan given by mouth. On the sixth day of her disease the patient died. A diagnosis of tetanus was made upon the clinical symptoms only. The bacteriological examination and the inoculation test upon animals both remained negative.

The Mechanical Sterilization of Rubber Gloves and Their Value in Practice.—This interesting subject receives attention from WANDEL and HOEHNE, in the *Münchener med. Wochenschrift*, 1903, No. 9. These observers found that vigorous washing of rubber gloves with soap and water sufficed to remove from the outer layers of the gloves bacteria. Simple washing with soap and water was not sufficient for this purpose. After the gloves have been used they should be washed while still upon the hands with soap and water. They should then be turned, again washed, and tried by filling them with fluid to detect a tear or perforation. They should then be powdered with sterile powder and kept in gauze or linen. Gloves so treated can usually be used five or six times.

Acute Contagious Pemphigus in the Newborn.—Before the Obstetrical Society of London, at its meeting on November 4, 1903, MAGUIRE read a paper upon this subject (*British Medical Journal*, November 14, 1903).

Most authorities consider pemphigus in the infant usually the result of syphilis. He reported the cases of eighteen infants suffering from acute contagious pemphigus, among whom eight deaths occurred. From the study of these cases, he concluded that a common contagion of unknown septic origin was conveyed from case to case by a certain midwife. The contagiousness was proved when the number of cases infected from each infant was ascertained. In two of the cases bacteriological examinations revealed the presence of the staphylococcus pyogenes aureus. The secondary symptoms were those of acute toxæmia, the infection having gained entrance through the unhealed umbilicus. The writer concludes that while this comparatively rare disease usually attacks the newborn, it may also be found in older

children and adults. The eruption was bullous, variable in distribution and extent, and the specific germ was found in the contents of the vesicles. In mild cases no other symptoms than the eruption were present. In grave cases there was a general infection, with an acute toxæmia, which invariably ended fatally. The unhealed umbilical scar gave access to the poison in these cases. No method of treatment employed influenced the course or termination of the disease.

In discussion, Cullingworth had seen a case in an adult when it was very difficult to recognize the source of infection.

Dickinson referred to an epidemic of fourteen cases in the Foundling Hospital at Parma. Most of these cases were fatal, and an examination of the blood showed a marked leukocytosis caused by this specific organism. Syphilis had nothing to do with it. He did not believe that infection through the umbilicus was the cause of the toxæmia, as cases in adults could not be explained in this way.

MacLeod had observed the increasing rarity of this disease, and believed it due to the general use of antiseptic precautions in maternity hospitals. He believed that the streptococcus was the cause of the infection. In determining the germ he has used the method of Sabouraud, of Paris, who aspirated the bullæ into a sterile pipet containing acetic fluid. By this means MacLeod obtained streptococci in pure culture in a fatal case. He referred to the close resemblance of the disease to impetigo, whose cause had been found to be streptococcus pyogenes. He described a case in which an abrasion of the skin at the lip had been the point for the entrance of infection. He thought that usually a diagnosis could be readily made between this disorder and syphilitic pemphigus. A form of hereditary pemphigus, the result of vasomotor disease, might occasion difficulty in differential diagnosis.

Gonorrhœa during Pregnancy and the Puerperal State.—Fruehinsholz (*Zentralblatt für Gynäkologie*, 1903, No. 45) finds that gonorrhœa occurs among pregnant women in from 20 per cent. to 25 per cent. of cases. The presence of an acute gonorrhœa does not prevent conception. Pregnancy causes latent symptoms of gonorrhœa to develop. Gonorrhœa does not, as a rule, bring on abortion, although, when abortion occurs in patients suffering from gonorrhœa, gonococci are often found in the decidua and placenta.

During the puerperal period gonococci increase rapidly in number, are found in the vaginal secretion, and often become abundant in the lochia. Puerperal infection is undoubtedly caused in some cases by gonococci. Cases of so-called puerperal rheumatism are most frequently the result of gonorrhœal infection.

Ectopic Gestation; Removal of Living Fœtus by Section, with Persistence of Secondary Hemorrhage —In the *Journal of Obstetrics and Gynecology of the British Empire*, November, 1903, Malcolm reports the case of a patient, aged twenty-three years, previously healthy, who was seized with severe pelvic pain, followed by vomiting, nausea, and shock. The pain soon passed away and was followed by irregular menstruation. A second attack of severe abdominal pain, with vomiting and nausea, occurred, followed by what was apparently a menstrual discharge, and shortly after this a short attack of pain and vomiting. After a fourth attack of pain a slight rise of temperature was observed.

The patient was examined two and a half months after the first irregularity of menstruation. There was a mass, about the size of a three months' pregnant uterus, in the pelvis, extending more to the right than to the left. The lower border of this mass was below the os uteri and very firm. The cervix was high up, close to the pubic bones, a little to the left of the median line. The body of the uterus was attached to the left side of the abnormal mass. Its cavity was three inches deep. The patient's color was sallow, the breasts showed no sign of pregnancy, and the pulse was good. Immediate operation was declined. After a fifth attack of pain, the abnormal mass had increased in size, and great tenderness was present. The temperature was 101.5° F.; the pulse was 90; the complexion more sallow. There was frequent vomiting. On operation a sac filled with pus was found outside the peritoneal cavity. This sac contained a living fœtus. The placenta had a large attachment upon the right side of the pelvis and was allowed to remain. The amniotic sac was washed out and packed with iodoform gauze. The patient did well after the operation until the fifth and sixth days, when, upon changing the packing, a very free hemorrhage occurred from the cavity, which was controlled by firm packing. Changing of the dressing was invariably followed by hemorrhage. Two weeks after the operation the placenta was seen in the wound looking healthy and evidently well nourished. The cord had sloughed away. An attempt to separate the placenta was followed by profuse bleeding. The second effort to remove the placenta succeeded in bringing away a small piece, followed by copious hemorrhage. After this hemorrhage ceased the placenta evidently died, suppuration increased, and the placenta had to be removed by the finger. This was accompanied by an offensive discharge, by prostration, and free sweating on the part of the patient. Gradually the temperature dropped, the discharge ceased, and the patient made a good recovery. The placenta had been adherent over the right side of the pelvis nearly as high as the brim, over a portion of the left side of the sacrum and the left side of the pelvic floor. After the removal of the placenta a drainage-tube was introduced into the sinus. The patient had occasional attacks of pain, with increase in fever, and passed mucous casts from the intestine. She finally made a complete and satisfactory recovery. It is probable that in this case the Fallopian tube burst into the cellular tissue between the layers of the broad ligament, the placenta becoming attached to the wall of the pelvis in such a position that it did not involve any portion of the intestine. The gestation sac gradually raised the peritoneum out of the pelvis. Hemorrhage into the tissues had not occurred at any time. This case is also interesting from the fact that it occurred in a woman previously perfectly healthy and soon after her marriage.

Twin Pregnancy in Utero, with Ruptured Tubal Gestation.—In the *Journal of Obstetrics and Gynecology of the British Empire*, November, 1903, MARSHALL reports the case of a patient who had had four children with easy labors and good recoveries. Recently she had suffered from pain in the lower abdomen, slight vaginal discharge, and occasional faintness. She considered herself three months pregnant. She had had several attacks of severe pain, with collapse, vomiting, and great thirst. On admission to the hospital the patient had evidently had severe hem-

orrhage. The temperature was subnormal and the pulse very rapid. The abdomen was much enlarged, with dulness over the lower part and in the flanks. An ovoidal tumor, the uterus, extended upward to within three inches of the umbilicus and deviated to the right. On the left side there was a cystic partially fixed tumor lying deep in the pelvis, and very tender to the touch. It was evident that intrauterine and extrauterine pregnancy were both present.

On operation the abdomen was found full of fluid blood. A large ruptured ectopic gestation sac was found on the left side of the pelvis, from which a well-developed three months' fœtus was removed. As hemorrhage was active, the ovarian artery was ligated; the placenta was removed without injury to surrounding organs. As hemorrhage could not be checked without ligating the ovarian and uterine arteries, the uterus was removed. The oozing from the sac practically ceased, and the cavity was firmly tamponed with sterile iodoform gauze, the ends being brought into the vagina. The abdomen was closed with silkworm-gut suture. Under free stimulation the patient reacted, and forty-eight hours after the operation the clamps, which had been placed upon the broad ligaments and the gauze packing, were removed. There had been no hemorrhage. The pulse remained fuller and better. Secondary collapse came on; the patient complained of pain in the abdomen; the pulse became rapid and feeble, and death followed.

Upon autopsy the wound was clean and there were no signs of peritonitis or hemorrhage. Death was due to excessive loss of blood. The uterus contained a twin pregnancy at three and a half months. In this case the fetal sac had evidently ruptured slightly on several occasions, the last and possibly the largest rupture being caused by an enema administered when the patient entered the hospital.

GYNECOLOGY.

UNDER THE CHARGE OF

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Disadvantages of Trendelenburg's Posture.—FRANZ (*Zentralblatt für Gynäkologie*, 1903, No. 32) after reviewing the cases reported by Kraske, Trendelenburg, and others, in which serious circulatory disturbances followed operations in the elevated position, states that in 745 abdominal sections he did not observe a single case. Studies of the pulse and respiration-curves led him to conclude that with the pelvis elevated the abdominal breathing is constantly diminished, while there is only a slight, if any, compensatory increase in the thoracic.

As regards the effect of anæsthesia, the writer noted in 825 cases of ether narcosis in the dorsal position 19 (2.3 per cent.) cases of bronchitis,

while in 493 in which the pelvis was elevated there were 44 cases (8.9 per cent.); 150 chloroform narcoses with the patient in the dorsal position were followed by 4 (2.7 per cent.) cases of bronchitis, and 233 in Trendelenburg's posture by 9 (3.9 per cent.), showing that with the latter anæsthetic the difference is much less marked than when ether is used. The writer explains the frequency of bronchitis after operations in the elevated position as due to the escape of the infection mouth-contents into the bronchi after the patient is lowered, and suggests the advisability of carefully cleansing the mouth before the change is made to the dorsal posture.

He further emphasizes the fact that the diminished oxygenation, which is undoubtedly present when the patient is in the elevated posture, probably influences unfavorably the central nervous system, and that many disturbances which are referred to opening the abdomen are really due to this cause, since they are more often absent when the vaginal route is adopted.

The Abdominal and Vaginal Routes.—ERYSTROM (*Zentralblatt für Gynäkologie*, 1903, No. 32) compares his mortality after abdominal section (2.6 per cent.) with Dührssen's after vaginal operations on the adnexa (2.8 per cent.), and draws the following conclusions: The results of laparotomy for diseased adnexa are as good as those following vaginal section, but the former operation is more severe and the convalescence is longer. While the abdominal wound may be the cause of subsequent complications, the vaginal is not seldom a source of trouble. By the abdominal route the exact nature of the lesion can be better ascertained and repaired. The writer would by no means abandon vaginal section, but he believes that each case should be carefully studied and the indications clearly noted.

Development of Cancer from Mucous Polypi.—OPITZ (*Zeitschrift für Geb. u. Gyn.*, Band xlix., Heft 2) reports two cases, the first of which presented unusual features. The patient, aged fifty-seven years, had passed the menopause seven years before. On account of persistent hemorrhage, exploratory curettement was performed and the diagnosis of adenocarcinoma was made. After removal of the uterus a polypus was found near the fundus that had undergone sarcomatous degeneration. Cancerous nodules were scattered over the endometrium, and the gland near the surface of the tumor presented the typical structure of adenocarcinoma. In the second case a polypus was removed from the cervix of a patient fifty-eight years of age who had had hemorrhages and a foul discharge for several months. As the growth proved to be cancerous, vaginal extirpation was performed subsequently. The tumor showed a connective-tissue stroma with large cancerous alveoli, the cells being continuous with the squamous epithelium in the surface. Normal glands were present between the alveoli.

Endothelioma of the Cervix Uteri.—KIRCHGESSNER (*Zeitschrift für Geb. u. Gyn.*, Band xlix., Heft 2) adds the following case to the eight already reported. The patient, a young multipara, had suffered for a year and a half with a bloody discharge. On examination a flat, irregular growth the size of a walnut was seen on the anterior lip of the cervix, the posterior lip being normal, and the uterus and adnexa

non-adherent. The patient died of peritonitis four days after removal of the uterus. Microscopically it appeared that the neoplasm had developed from the endothelium of the lymphatics and lymph spaces of the portio.

Etiology of Adenocystoma of the Ovary.—WALTHARD (*Zeitschrift für Geb. u. Gyn.*, Band xlix., Heft 2) as the result of his studies of serial sections of eighty normal ovaries from subjects of all ages, rejects the term "germ-epithelium" in favor of ovarian or surface-epithelium. In the ovaries of the newborn the follicular epithelium appears sometimes in solid masses and sometimes in the form of glandular pouches. The latter may develop into the primary follicles or may persist in the form of pouches or retention-cysts lined with flattened cells.

The writer believes isolated groups of cells found throughout the stroma subsequently form the surface epithelium. These cells may at any time after birth form glandular ingrowths from the surface, or may give rise to adenomatous growths within the stroma. Small collections of pavement epithelium are seen in the stroma or on the surface of the ovary which may separate in their centres and then form cysts .

Similar islands of ciliated epithelium among the surface cells may give rise to pouches lined with ciliated cells. Similar pouches are formed by masses of cubical cells, which are sometimes seen in the stroma as well as on the surface, and appear to be entirely independent of the ovarian epithelium. In all these pouches it is possible to trace two processes, either degeneration or proliferation, to the glandular type.

The remains of the primordial kidneys can be readily recognized in the hilum. The canals are greatly dilated and are lined with flattened epithelium. The writer was never able to find any trace of adenomatous structures in these. The pavement, ciliated, and cubical (or goblet-shaped) cells in the ovary are not derived, he thinks, from the surface or follicular epithelium, or from the remains in the hilum, but they are to be regarded as congenital cell-nests.

Treatment of Inversion.—FRESSON (abstract of monograph in *Annales de Gynécologie et d'Obstétrique*, October, 1903) lays down the following rules: 1. Manual reduction should be attempted in every case of inversion, whether acute or chronic. 2. Bilateral incision of the cervix is applicable to all acute cases, and to those chronic cases in which the obstacle to reduction is demonstrated to be a constricting ring. 3. In others colpohysterotomy is the operation of election. 4. Vaginal hysterectomy is indicated only in the presence of intractable hemorrhage, infection, or when the irreducible uterus has been so injured that there is no probability of its regaining its functional integrity. 5. Hæmisection is the most rapid and convenient method of extirpating the organ.

Vulvovaginitis in Children.—BERKENHEIM (*Wratsch; Zentralblatt für Gyn.*, 1903, No. 32) reports the results of observations extending over ten years, from which he infers that 75 per cent. of the cases of vulvovaginitis in little girls is due to gonorrhœa. The relatively mild course of the inflammation is due to the shortness of the vagina in children, the absence of folds and glands, and the slight development

of the vessels. The absence of such disturbing factors as coitus, menstruation and pregnancy favors the rapid cure. The average duration of the affection is eight weeks, but one-third of the cases become chronic.

Painful urination, local hyperæmia and leucorrhœa are the common symptoms. Complications are rare, especially peritonitis, urethritis, cystitis; arthritis and conjunctivitis are more common. Arthritis generally affects single joints, and the prognosis is favorable.

The writer recommends vaginal injections of solutions of sulphate of zinc, boric acid, permanganate of potash, protargol, and ichthyol, although he has never seen a case aborted by the use of these remedies. It is important that the patient should not be allowed to use the same vessels or towels as the other children.

The Normal and Diseased Peritoneum.—CLAIRMONT and HABERER (*Wiener klin. Wochenschrift*, 1902, No. 45) conducted a series of experiments on rabbits to determine the absorptive power of the peritoneum. He followed the suggestion of Schnitzler and Ewald, injecting a 2 per cent. solution of iodide of potassium and noting its appearance in the urine. He adduced the following: Increased peristalsis increases absorption, while the introduction of air has no effect whatever. Sterile urine or intestinal contents do not, as a rule, hinder absorption.

In the early stage of peritonitis peritoneal absorption is increased, but later it is diminished. The latter result is also observed after abdominal sections in which dry sponging is employed, the converse being true when the wet method is adopted. In general, absorption is differently affected by different anæsthetics; transudation is not much affected by laparotomy.

A marked diminution of the power of absorption is noted after removal of the peritoneal covering of the diaphragm.

Treatment of Hæmatocele.—BONGLE (*Arch. gén. de Méd.*, 1903, No. 3) believes that many small retrouterine hæmatoceles are absorbed without treatment. Large circumscribed ones may be incised and drained per vaginam, especially if suppuration is suspected. If it is inferred that several small collections are present, especially if the patient's menstrual period is due, vaginal incision may be attended with profuse hemorrhage, requiring immediate abdominal section. In other cases, in fact in the majority of hæmatoceles, laparotomy is the preferable procedure, above all when active bleeding is suspected.

Pigmentations of Genital Origin.—DALCHE and FOUQUET (*La Gynécologie*, 1903, No. 2) believes that certain pigment changes in the skin (chloasma, vitiligo, etc.) are of reflex uterine or ovarian origin, possibly due to disturbances in the so-called internal secretion of the ovary. They recognize several varieties, viz.: pigmentation attending menstrual disturbances, that due directly to organic utero-ovarian disease, and a third form which appears in connection with chlorosis, Raynaud's disease, etc., which may also be referred to some pelvic trouble.

As regards local treatments the authors recommend various ointments. The general treatment consists in the relief of amenorrhœa and dysmenorrhœa, or the removal of diseased organs. Oötherapy is also advised.

OPHTHALMOLOGY.

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Corneal Ulceration Treated by Exclusion of Actinic Light.—W. LOWE (*Intercolonial Medical Journal of Australasia*, March 20, 1903) reports a case of ulceration of the cornea of two years' duration treated by exclusion of actinic light. The patient had intense intolerance of light, and was confined to a darkened room. Every form of treatment was tried without avail, until, acting on the theory that the actinic rays were keeping up the trouble, the patient was placed in a room the windows of which were covered with red photographic tissue paper. The effect was most remarkable. The intolerance to light and pain disappeared, while the injected cornea blanched as if adrenalin had been applied. The next morning lacrymation and discharge ceased, and on the third day the patient could read and sew. No sign of ulceration could be seen. The next day she went out of doors protected with ruby glass goggles.

The writer thinks that the red rays may have some germicidal action, or they may act by allaying inflammation.

Glaucoma.—WAHLSFORS (*Arch. of Oph.*, November, 1903) insists that the conception of glaucoma, which goes back to von Graefe as being one with increased tension, is false. The attempt to differentiate certain cases of so-called simple glaucoma without increased tension from true glaucoma is a mistake. They are both essentially the same disease. We see simple glaucoma pass into inflammatory, and *vice versa*. He argues that the increase in tension is something accidental, and not even essential to glaucoma.

As regards the opinion now largely prevalent, that the cause of glaucoma lies in closure of the channels of exit, the writer asserts that while this may play an important role in inflammatory glaucoma, it is not applicable in simple glaucoma, where the increase in tension is quite subordinate and sometimes even absent. The latter class is very important in the explanation of glaucoma, as showing that this disease may exist without tension and without retention, thus proving that retention is not the cause of glaucoma. The writer calls attention to the variations in the fields. The defect may be central, peripheral, or irregular, or sector-like. There is no typical glaucomatous field. Excavation appears as a rule as a late sign. He insists upon the importance of one symptom as a true indication of the nature of the fundamental cause. This is reduction of the light-sense first, noticed by Forster, and emphasized by Mauthner. Hemeralopia may be the first and only

symptom present for a long time, perhaps even overlooked by the patient, if confined to the periphery. The cause of hemeralopia is functional disturbance of the rods and cones in consequence of imperfect nutrition, the cause of which must lie in the inner layers of the choroid, whose vessels supply the outer layers of the retina. In the choroid, then, we must seek the cause of simple glaucoma. The choroidal lesion is an atrophy. The functional disturbances in simple glaucoma—diminution of the light-sense, narrowing of the field of vision, and diminution of central vision—may readily result from the atrophic process, which does not at once extend over the entire choroid, but begins usually in the periphery and gradually extends toward the centre, although the reverse is sometimes observed. The sector-like defects of the field depend upon limited layers of atrophy. The excavation of the nerve head is also best explained when the resistance is diminished by preceding inflammatory process, such as sclerochoroiditis. A normal disk possesses sufficient resisting power to withstand not only the normal tension, but considerable increase, as may often be observed in adherent leukoma, where we find a normal disk, with considerable increase in tension for years. Where the tissues of the disk (lamina cribrosa, in the formation of which the choroid takes some part) have lost their resistance, excavation may come on independent of increased tension, which should, therefore, not be designated pressure excavation.

The increased tension may also be explained as the result of the choroidal atrophy. Normally the choroid exerts pressure upon the liquid contents of the eyeball, as is shown by Donders' observation, that when all the coats of the eye are divided together the choroid retracts from the margin of the wound and leaves the sclera exposed. This contractility is doubtless due to the numerous muscular fibres which accompany the larger vessels and to Bruecke's muscle, which acts as a tensor. Such a muscular network acts as a restrainer of tension, not only by its direct resistance to the pressure of the contents, but also increases the lymph circulation (the currents of liquids) through the pressure it exercises. In glaucomatous increase of tension three factors are at work—paralysis of the muscular network of the choroid, causing a slowing of the currents of liquid; the channels of exit in consequence of the retarded flow become blocked and cause a retention of the ocular liquids, and finally, the venæ vorticosæ, which are compressed by the increased tension, lead to a venous stasis with all its results. As the disease process is due to choroidal atrophy, the prognosis of glaucoma in general must be considered bad, as we have no remedies to act on this process. The writer thinks that repeated injections of strychnine prevent the spread of the atrophy. He believes that an injection per day for ten or twenty days, repeated three or four times a year, has enabled him to preserve the vision for many years in simple glaucoma, with little or no increase in tension.

In simple glaucoma iridectomy is useless as a curative, but valuable as a prophylactic measure against acute attacks. Where the tension is increased iridectomy remains the sovereign means of reducing it, but its action is not understood; Exner's explanation that the short arterial twigs proceeding from the major arterial circle of the iris form direct anastomoses with the corresponding venous twigs, without having to pass through the capillary network, is probably the best. Iridectomy thus works, as Knapp expresses it, as a safety-valve. As,

however, the atrophic process continues, central vision becomes poorer, the field contracts, and the disk becomes excavated.

Sclerotomy, both anterior and posterior, as long as the wound is not thoroughly healed, is useful in giving time for the circulatory disturbance to pass off. Eserine reduces tension in fresh cases of acute congestive glaucoma, through causing contraction of the tensor of the choroid and probably of its muscular network. In simple glaucoma eserine is only valuable so long as the atrophic process has not entirely paralyzed the muscular network and the tensor.

Some Modern Views on Primary Glaucoma.—ELLIOT, I. M. S. (*Ind. Med. Gaz.*, July, 1903), gives a résumé of the above subject, based on a review of the opinions of leading authorities as found in the literature of the subject, as well as from personal interviews. He sums up that under the term simple glaucoma have been in the past included: (1) cases in which the prime factor was disease of the optic nerve; (2) cases in which the triad of symptoms alone occur—*i. e.*, cupped disk, retracted field, and diminution of visual acuity, and (3) a long series of cases passing insensibly from the last class and merging at the opposite end of the scale into well-marked congestive glaucoma. Atrophic cases should be carefully distinguished and placed apart. Classes 2 and 3 may be separated from each other by advancing knowledge, which may lead to a better comprehension of their pathology, a result greatly to be desired, establishing more exact indications for operation. As respects treatment of glaucoma, there is no divergence as to the rational and medical measures—myotics and general hygiene.

Of surgical operations, sclerotomy and iridectomy, the writer concludes that the modern ophthalmologist undertakes iridectomy with a much more expectant heart than that with which he performs sclerotomy. Many operators prefer sclerotomy (1) in early cases where there is considerable doubt, (2) in late cases where it is employed as a *dernier ressort*, (3) in desperate cases where iridectomy has failed, and (4) in simple glaucoma where any operation is doubtful, and, therefore, the procedure of least magnitude is chosen, although the balance of safety does not lie wholly with the latter operation. Sympathectomy finds its rôle according to Abadie, in those cases in which the triad alone is present, which do not yield to medicinal and rational treatment.

The Wearing of Artificial Eyes over an Eyeball in Situ and after Abscission of Staphyloma.—F. A. Müller's Sons (*Klin. Mon. f. Augenheilk.*, October, 1903) call attention to the impracticability of fitting the ordinary shell, which answers well enough in cases like phthisis bulbi, to the full eyeball, or such as are but slightly shrunken, the thickened spot convex posteriorly in the region of the iris of the shell, exerting undue pressure if applied to a full globe and preventing perfect apposition. The perfectly flat shells still found in the shops are entirely worthless. They propose to meet the cases in question by giving the prosthesis a smooth concavo-convex form to permit perfect apposition.

The writers also criticise the horizontal cicatrix with its angular edges, which results from the usual mode of abscising staphylomata (a horizontal elliptical section with vertical sutures). These edges enormously increase the difficulty of prosthesis and quite destroy the cosmetic effect,

for the reason that the bridging of these edges requires the shell to be broader horizontally, so that the artificial eye seems longer and larger than the sound one. To obviate this the writers raise the question whether the wound of excision for the staphyloma should not be given a vertical direction. The edges would then come to be beneath the lids, at which points the shell could be easily cut out to correspond; the horizontal meridian, which is the most important, would then furnish a stump of suitable convexity. A more symmetrical result might conceivably be secured by a triangular section with a purse-knot suture. The writers do not venture to offer an opinion whether this is a practicable procedure.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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The Reactions of the Blood in Experimental Diabetes.—SWEET (*Journal of Medical Research*, 1903, vol. v. p. 255) says the fact long known to clinicians and students of immunity, that the diabetic organism is abnormally susceptible to infectious processes suggested the experiments which are the subject of this communication. Studies were made upon the hæmolytic and, to a less extent, the bactericidal power of the serum of rabbits suffering from phlorizin and adrenalin glycosuria, and of the serum of dogs which were the subjects of true diabetes mellitus caused by total extirpation of the pancreas. After subcutaneous injections in rabbits of an alcoholic solution of phlorizin, the serum showed a slight though readily demonstrable increase in the hæmolytic complement for bovine erythrocytes. This increase in complement is to be explained as an occurrence coincident with the inflammatory reaction of the organism to the injection. Such injections of phlorizin produced no effect upon the amboceptor for bovine erythrocytes. Intraperitoneal injections of adrenalin chloride were followed by much the same results as regards the serum reactions of the rabbits as was noted in the experiments with phlorizin. The only change exhibited by the serum was a slight increase in the hæmolytic complement, which occurred in certain animals that developed an inflammatory reaction following the intraperitoneal injections. Although injections of both phlorizin and adrenalin chloride give rise to a glycosuria, the condition cannot be considered as a diabetes mellitus, and therefore to reproduce this disease in animals as nearly as possible excision of the pancreas was resorted to; dogs were used for this purpose. Fourteen dogs were operated upon, and of this number two recovered completely from the operation, dying subsequently of a true diabetes mellitus. Following

upon the diabetes produced by complete removal of the pancreas, the hæmolytic activity of the dogs' serum was markedly decreased for both rabbit and guinea-pigs' erythrocytes. There is, besides, what was interpreted as a complete loss of the normal bactericidal property of the serum for such bacteria as B. coli communis, B. typhi abdominalis, and B. dysenteriæ, while somewhat less conclusive was the decrease of bactericidal power of the blood for staphylococcus pyogenes aureus, since the serum of the normal dog has very little if any bactericidal effect upon this organism. The decrease in the hæmolytic action of the serum of the diabetic dog was, furthermore, shown to be due to a loss of hæmolytic complement, and from analogy the loss of bactericidal power was believed to be dependent upon the loss of bacteriolytic complement. Unless the pancreas was completely removed the above phenomena did not occur; consequently the author believes that extirpation of the entire organ is as necessary for the loss of complement as it is for the production of diabetes. In diabetic dogs the organism had not entirely lost its power of reacting to inflammatory processes by an increase of complementary substances. With these changes in the complements no disturbance of the normal relation of the receptors of the erythrocytes to specific hæmolytic amboceptors could be demonstrated. The author believes that the loss of the complementary substances in diabetes mellitus points conclusively to the fact that there is no relation between the leukocytes of any type and the production of the complements. During the experiments it was discovered that secondary infections were not accompanied by a decrease in the glucose excreted by the diabetic organism.

Contribution to the Physiological Study of Glycerin.—NICLOUX (*Journ. de Phys. et de Path. Gén.*, 1903, T. v. p. 527) has found that under normal conditions glycerin is present in the blood of dogs and rabbits, 2 to 2½ mgr. of glycerin occurring in 100 c.c. of blood from dogs and 4 to 5 mgr. in rabbits' blood. The ingestion of fats does not appear to alter the proportion of glycerin in the blood. After an intravenous injection of glycerin this substance disappears rapidly from the blood and the relative proportion of glycerin in the urine is much increased above the amount found before the intravenous injections. Since the same relative increase of glycerin occurs in the urine after ingestion of glycerin, the author believes that the cells of the kidney possess a peculiar selective function for the excretion of this substance.

The Experimental Production of Uncompensated Heart Disease, with Especial Reference to the Pathology of Dropsy.—CHARLES BOLTON (*Journal of Path. and Bact.*, 1903, vol. ix. p. 67) says three distinct theories exist at present to explain the primary factors concerned in the pathology of the dropsy of venous stagnation: 1. A secretory theory, of which Hamburger is the chief exponent, and according to which the capillary endothelial cells are believed to have a specific secretory function increased in dropsy. 2. A theory originated by Lazarus-Baslow which explains the œdema by assuming that an accumulation of waste products occurs in the tissues. 3. A mechanical and physical theory, according to which the œdema depends upon increased venous and

capillary pressure, together with an alteration in the vessel wall. Cohnheim and Starling are among the main supporters of the mechanical theory. Cohnheim found that when oil was allowed to flow into the pericardial sac the general venous pressure was raised and the arterial pressure lowered, thereby causing increased capillary pressure with diminished velocity of blood stream. Bolton, pursuing somewhat the same line of investigation, but modifying the experiments, brought about an interference with the diastolic filling of the heart by constriction of the pericardium of cats with the aid of a line of sutures. Following this operation the animals rapidly developed effusions into the pleural and peritoneal cavities and showed some subcutaneous œdema. By this operation, therefore, one of the sequences of uncompensated heart disease could be successfully reproduced. In a second group of experiments the pericardium was constricted and an estimation made of the blood pressure in the peripheral arteries as well as in the inferior vena cava, portal vein, and peripheral veins before and at different periods during and after the operation. Upon constricting the pericardium there resulted immediately a considerable decrease in the arterial pressure and a rise of pressure throughout the entire venous system; but, whereas the arterial pressure remained low for some hours after the operation, the venous pressure gradually fell to normal. If the constriction was now relieved the arterial pressure rose to normal or rose slightly above normal, while the venous pressure dropped below normal. By intravenous infusions given while the pericardium was constricted the arterial pressure could not be elevated to its normal range, nor could the high level of the venous pressure be maintained. Cohnheim's experiments were repeated, and it was shown that when the pericardium is distended with oil the heart gradually tends to fail owing to the lack of blood supply, which is cut off by the pressure of the oil upon the thin-walled inferior and superior vena cava at their entrance into the auricle. Thus the condition brought about by Cohnheim's experiment is essentially different than that of the author's experiment, where the action of the heart as a force pump alone is interfered with and conditions are much the same as are produced by an adherent pericardium. Finally a third group of experiments demonstrated that when the animals were allowed to live after constriction of the pericardium until the onset of œdema, the arterial pressure gradually rose but never reached the normal level, while the venous pressure remained stationary. Bolton concludes that in uncompensated heart disease, such as is imitated by the present experiments, the capillary pressure is of necessity low, and he agrees with Starling in adopting the mechanical theory as an explanation for the development of œdema.

Leukæmia.—HANS LUCE (*Deut. Arch. f. Klin. Med.*, 1903, Bd. lxxvii. p. 255) records a peculiar form of lymphatic leukæmia for which he has suggested the name of "leukanæmia." A woman, aged forty years, developed a simple anæmia following an attack of acute tonsillitis. After six months' time the blood picture changed and assumed the characters of a typical lymphatic leukæmia. The anæmia increased with the appearance of a hemorrhagic diathesis until at the end of three and a half months the histological blood picture was typical of both lymphatic leukæmia and pernicious anæmia. For two months preceding the patient's death this condition remained more or less

constant. At autopsy all the organs showed an extreme grade of anæmia, while an extensive fatty degeneration was noted, especially of the heart muscle and liver. The bone-marrow was red. Microscopically there was leukæmic infiltration of the liver, kidneys, and heart muscle, with lymphadenoid hyperplasia of the spleen and cervical lymph glands, and almost complete lymphadenoid metaplasia of the bone-marrow. But the bone-marrow showed, besides, the histological elements characteristic of pernicious anæmia. The complete absence of siderosis throughout all the organs examined was noteworthy. This case together with those of Körmiöczi and Leube form a small group for which the author believes the term leukanæmia suitable—instances where the blood pictures of lymphatic leukæmia and pernicious anæmia are combined. The condition is looked upon as a disease of the bone-marrow, and the anæmia is considered to be of myelogenic rather than hæmatogenous origin, a view which is strengthened by the fact that there was no pigment in any of the organs. It has been shown that in certain cases of lymphatic leukæmia the lymphoid tissue of the bone-marrow is the seat of extensive hyperplasia. In instances such as the present it is probable that the lymphoid metaplasia takes place at the expense of the erythroblastic tissues, and thus there results an anæmia comparable in many respects to the aplastic forms of pernicious anæmia described by Ehrlich.

A New Cholera Vaccine, and its Method of Preparation.—Strong (*Amer. Med.*, 1903, vol. vi. p. 272) has found that he can produce a cholera vaccine which contains free receptors, and has the power of building both uni- and amboceptors (agglutinins and bacteriolysins) in a cholera immune serum, by autolytic digestion of cholera spirilla in aqueous solution. When injected into rabbits this vaccine gives rise to the appearance of bactericidal and agglutinative substances in the blood sera of these animals which equal or exceed those obtained from inoculations of virulent living cholera vibrios. One advantage gained by the use of the vaccine instead of the living or dead bacteria is the absence of local reaction following the inoculation. The vaccine filtrate may be evaporated to a powder which when redissolved in water and injected into animals is capable of giving rise to an immunity. The author suggests that free receptors obtained by autolytic digestion and filtration of other bacteria may be used as a vaccine in diseases such as typhoid fever and dysentery.

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CONTENTS.

ORIGINAL COMMUNICATIONS.

PAGE

Gastroenterostomy. By JOHN B. DEAVER, M.D. 187

The Nervous Complications and Sequelæ of Smallpox. By CHARLES J. ALDRICH, M.D. 198

A Case of Typhoid Fever Presenting an Arterial Complication—Probably an Arteritis. By WALTER R. STEINER, A.M., M.D. 212

The Occurrence of Cells with Eosinophile Granulation and their Relation to Nutrition. By EUGENE L. OPIE, M.D. 217

Circumcorneal Hypertrophy (Vernal Conjunctivitis) in the Negro. By SWAN M. BURNETT, M.D., PH.D. 239

A Case of Seborrhœa Nigricans (Black Mask of the Face). By ARTHUR VAN HARLINGEN, M.D. 245

A Report of Two Cases of Ectopic Gestation and a Case of Intestinal Obstruction Due to Meckel's Diverticulum. By HAROLD H. HEYER, M.D., and H. M. LEE, M.D. 248

Ludwig's Angina. Report of a Case. By GWILYM G. DAVIS, M.D. . . . 263

A Fatal Case of Poisoning with Oil of Gaultheria. By J. WOODS PRICE, M.D., and EDWARD M. L'ENGLE, M.D. 265

The Significance of Urinalysis in Pregnancy, with Especial Reference to Eclampsia. By ROBERT N. WILLSON, M.D. 267

A New Method of Gastric Proteolysis. By A. L. BENEDICT, A.M., M D. . 274

Thrombosis of the Midcerebral Artery Causing Aphasia and Hemiplegia. By CHARLES W. BURR, M.D. Remarks on Cerebral Skiagraphy. By G. E. PFAHLER, M.D. 282

Report of a Case of Tumor of the Brain, an Endothelioma of the Dura. Operation. Recovery. Death in 108 Days from Recurrence of the Disease. By WILLIAM J. TAYLOR, M.D. 287

A Report of Five Cases of Tumor of the Brain, with Operation. By WILLIAM G. SPILLER, M.D. 293

A Discussion of the Surgery of Tumors of the Brain, with a Résumé of the Operative Records of Four Craniotomies. By CHARLES H. FRAZIER, M.D. 316

REVIEWS.

Diseases of the Skin: Their Description, Pathology, Diagnosis, and Treatment, with Special Reference to the Skin Eruptions of Children, and an Analysis of Fifteen Thousand Cases of Skin Disease. By H. Radcliffe-Crocker, M.D. 331

PAGE

Nurses' Guide to Surgical Bandaging and Dressings. By William Johnson Smith, F.R.C.S. 332
Hyperæmia as a Therapeutic Measure (Hyperamie als Heilmittel). By Prof. August Bier 333
High-frequency Currents in the Treatment of Some Diseases. By Chisholm Williams, F.R.C.S. 335
A Text-book of Diseases of Women. By Barton Cooke Hirst, M.D. . 336
Gynecology. A Text-book for Students and a Guide for Practitioners. By William R. Pryor, M.D. 339

PROGRESS OF MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

AND

W. S. THAYER, M.D.

A Case of Pentosuria 343
The Influence of the Concentration of the Urine upon the Outcome of the Reactions for Albumin 344
Concerning the Diagnosis of Chronic Nephritis 345
Concerning the Absence of Casts from the Urine of Patients with Nephritis 345
Is Sugar Destruction Completely Stopped after Extirpation of the Pancreas? 345
Leukæmia and Tuberculosis 346
Arterial Thrombosis of Gonorrhœal Origin 346

SURGERY.

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D.,

AND

F. D. PATTERSON, M.D.

The Operation of Gastroenterostomy, with Indications for its Performance 347
Splenectomy for Banti's Disease 350
Adrenalin in Local Anæsthesia 350
Retroduodenal Choledochotomy 351
Ogston's Operation for Clubfoot 351
A New Modification of Maydl's Operation for Congenital Ectopy of the Bladder 352
Wound of the Epigastric Vessels as the Result of Puncture for Ascites . 352
Adenoma of the Umbilicus 352

THERAPEUTICS.

UNDER THE CHARGE OF

REYNOLD WEBB WILCOX, M.D., LL.D.

	PAGE
Colloidal Silver	353
The Abortive Treatment of Gonorrhœa	353
Hedonal	354
Phosphorus in Psychasthenia	354
Lecithin	355
The Treatment of the Cardiac Complications of Rheumatism	355
Arsenous Acid in Cancerous Ulcerations	355
Empyroform	356
The Action and Therapeutic Application of Radium	356
Ascites Treated by a Diet Free from Chlorides	356

PEDIATRICS.

UNDER THE CHARGE OF

LOUIS STARR, M.D.,

AND

THOMPSON S. WESTCOTT, M.D.

Infections of the Newborn	357
The Overlying of Infants	358

GYNECOLOGY.

UNDER THE CHARGE OF

HENRY C. COE, M.D.,

ASSISTED BY

WILLIAM E. STUDDIFORD, M.D.

Examination of the Blood in Cases of Ovarian Cyst	359
Cystitis in the Female	359
Plastic Operation for Hydronephrosis	360
Paraffin Injections in Incontinence of Urine	360
Operations for Uterine Fibroid	360
Myoperithelioma	360
Retroflexion	361
Treatment of Inoperable Cancer of the Uterus	361
Epidural Injection in Enuresis	362
Streptococci in the Normal Urethra	362
The Operative Treatment of Cancer of the Uterus	362

OPHTHALMOLOGY.

UNDER THE CHARGE OF

EDWARD JACKSON, A.M., M.D.,

AND

T. B. SCHNEIDEMAN, A.M., M.D.

PAGE

The Use of a Mydriatic after the Age of Forty-five 362
Periscopic Lenses 363
Intraocular Lipæma 363
Examination of 4608 Railroad Employés for Acuity of Vision, Hearing, and Color Perception 363

OTOLOGY.

UNDER THE CHARGE OF

CLARENCE J. BLAKE, M.D.,

ASSISTED BY

E. A. CROCKETT, M.D., P. HAMMOND, M.D., E. DE W. WALES, M.D., AND W. A. LE COMPTE, M.D.

Ueber Horubungen Mittlest des Phonographen 364
The Pathological Anatomy of the Temporal Bone 364
Ueber den Einfluss des Telephonierens auf das Gehoerorgan . . . 365
Recent Theories on Sound Conduction 365

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

SIMON FLEXNER, M.D.,

ASSISTED BY

WARFIELD T. LONGCOPE, M.D.

The Dwarf Tapeworm (Hymenolipsis Nana) 367
Localization of the Pneumococcus 368
A Delicate Test for the Demonstration of Bile Pigments in Urine . . 368
The Role of the Omentum in the Course of General Infections . . 369
An Experimental Study of Nephrotoxins 369

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

CHARLES HARRINGTON, M.D.

Effects of Compressed Air on the System 370
Factors which Determine the Local Incidence of Fatal Infantile Diarrhœa 371
The Relation of Sulphur in Illuminating Gas to Air Vitiation . . 372
The Relationship of Human and Bovine Tuberculosis 373
Prophylaxis of Malaria 374
The Use of Chemical Preservatives in Foods 374